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KIDNEY HOMOTRANSPLANTATION IN CASTRATED AND NONCASTRATED MALE GOATS*

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For any animal except man,[3, 6, 7, 9, 12, 13] the longest reported period for homotransplanted kidneys to secrete urine was in the castrated male goat[9] in which urine was secreted for a mean of 15.6 days.

This study was undertaken to determine what effect, if any, the absence of the male gonad has on the fate of a homotransplanted kidney. Kidneys were transplanted from normal bucks to normal bucks, castrated males to normal bucks, and normal bucks to castrated males for comparison with those from castrated males to castrated males,[8] to determine whether there is any difference in the fate of the homotransplanted kidneys among these four combination groups.

METHODS AND MATERIALS

Male American Angora goats, 4 to 6 years in age and 34 to 57 kg. in weight, were used. Although not inbred, they were the progeny of registered or carefully bred stock and were, in external appearances at least, fairly homogeneous. The castrated males had been castrated before the sexually mature age of 3 or 4 months; they were smaller than the bucks in body weight and horn structure, but no different in kidney or adrenal morphology.

Bilateral nephrectomy on the proposed host was followed in 20 hr. by preparation of the recipient site in the neck. Blood flow to the transplant was interrupted and after 24 to 50 min. was established on the host's carotid-jugular circulation. The end of the ureter was brought through an opening in the skin and kept patent thereafter with a short polyethylene tube.

Postoperatively the goat was tied by its horns to a crossbar designed to govern movement and thus protect the transplant. Each goat was offered formulated feed and roughage, and treated daily with penicillin and streptomycin. Progress notes included daily rectal temperature and the analysis of urine made to drip for 1 hr. into a wide mouthed plastic bag (the most satisfactory of several methods) and, each week, the value of whole blood nonprotein nitrogen or blood urea nitrogen.

The end point was oliguria of the transplant, less than 50 cc. within 24 hr., or death of the host. Bacteriologic cultures were made of most transplants. From all sacrificed goats the transplants were placed into formalin within 15 min. Sections from all transplants were stained with hematoxylin and eosin; sections from many were stained for iron, and by the periodic acid Schiff method, the Brown and Brenn method and with methyl green-pyronin-orange G.[14] The ipsilateral jugular lymph nodes, spleen, liver, heart, lungs, adrenals and testes were examined grossly and, frequently, microscopically. Control sections were examined from normal kidneys; and from one kidney, 27 days after autotransplantation.

RESULTS

No significant differences in any regard (table 1) were found among the four donor-host combination groups which will be described, there-

This study was begun at the Biophysics Division, Chemical Warfare Laboratories, Army Chemical Center, Maryland, and completed at the Medical College of Georgia.

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TABLE 1

*Results of kidney homotransplantation experiments**

Donor-Host Combination	No. of Experiments	Days of Function	
		Range	Mean
Castrated male to castrated male†	9	8–27	15.6 ± 6.8
Normal buck to normal buck	3	16–26	22.0 ± 5.4
Castrated male to normal buck	6	13–26	18.2± 5.0
Normal buck to castrated male	8	9–31	18.3± 8.4
Total	26	8–31	17.8± 6.9

* The six technical failures are not included here.

† Data on this group constituted preliminary report.[8]

fore, as one population. The 26 transplants (an additional 6 were technical failures and were excluded) secreted urine for a mean of 17.8 ± 6.9 days.

The function of the transplant varied the 1st week, but thereafter deteriorated. The specific gravity of the urine decreased early in the postoperative course. Protein values of 3+ or 4+ were found for many days in the urine from all transplants near the end point, and actual gels of clotted plasma (electrophoretic pattern of urine from the 2 transplants which were tested was found to be that of serum) were found for many days in the urine from many transplants. The daily volume decreased gradually from values over 500 cc. to less than 50 cc. Oliguria of the transplant was the end point for 20 experiments.

With the usually relentless uremia the condition of the host deteriorated. Fever was sustained in almost every host for several days, between the 4th and 11th days. At this time,

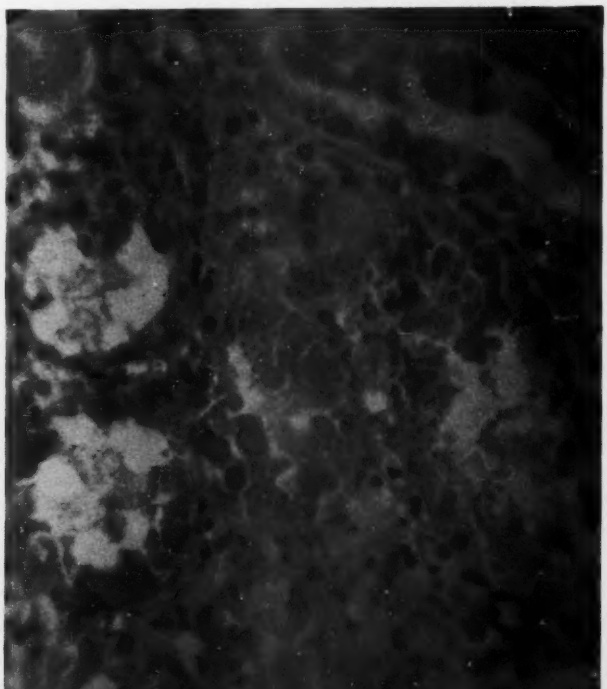

FIG. 1. Hyaline droplets in epithelial cells of convoluted tubules. Hematoxylin and eosin, × 650.

FIG. 2. Convoluted tubule with intact brush border (homotransplant secreted urine for 26 days). Hematoxylin and eosin, × 650.

weakness, anorexia, weight loss and frequently diarrhea presented and thereafter increased. Death of the host was the end point for 6 experiments; in 3 of these 6 hosts autopsy revealed severe pulmonary edema.

At autopsy, the weight of the edematous transplant had increased in range from 43–110 to 114–456 gm. The capsule was thickened and adherent to the neck tissues. Cortical ischemia was frequently severe; medullary congestion was variable, and hydronephrosis was minimal. Pyelonephritis was not obvious except in 3 cases although bacterial growth usually resulted from cultures. The renal artery and vein were patent.

Microscopic examination revealed that the edema involved all components. Mononuclear cells were numerous: the majority were small lymphocytes; many, however, in which the cytoplasm was stained with pyronine indicative of high ribonucleic acid content were recognized as plasma cells and so-called "immature plasma cells."[2] The cells were scattered in the interstitium and concentrated around the glomeruli, vascular poles of the glomeruli, arterioles and small arteries. They were not, however, within the muscular or subendothelial layers of the arteries. Furthermore, the lumens of the arteries appeared normal and, except in 1 case, were not encroached upon from swelling or proliferation of the endothelium. No pyroninophilia of the endothelium was found. Precipitated protein was abundant in the spaces of Bowman, as casts in the lumens of tubules and as droplets in the cytoplasm of the tubular cells (fig. 1), and was particularly abundant in those transplants which had secreted urine containing large amounts of serum. The glomeruli, except for this evidence of abnormal porosity, and a very slight, if any, proliferation of the cells of the tufts, appeared normal. Atrophy of the tubules was mild in most transplants (fig. 2); actual necrosis was present only, as a rule, in the subcapsular area.

Macrophages engorged with red blood cells were found in the venules and veins of many transplants, and in the medullary cords of the ipsilateral jugular lymph nodes of the hosts. Hemosiderosis was evident in the spleens. In the medullary cords of the ipsilateral jugular lymph nodes there were many mature plasma cells with pyroninophilic cytoplasm.

DISCUSSION

Use of castrated male goats as donors, hosts or both, in place of bucks caused no apparent difference in the fate of the kidney homotransplants in this study. Similar negative results were obtained in inbred mice with no difference being found between castrated and noncastrated males as hosts to skin isografts from male donors.[5] The test conditions of this study with inbred mice were prompted by the phenomenon reported by Eichwald and Silmser,[4] that C57Bl female mice eventually reject male skin isografts. Interestingly, of those females masculinized by castration and androgen injections, some did not reject the male skin as quickly. The preponderance of evidence indicates that the phenomenon is due, however, not to a lack of adequate androgens in the female host, but rather to a lack of a Y-linked histocompatibility gene. In rats, on the contrary, skin homografts fared better in those male hosts castrated immediately after birth according to Schäfer,[10] who postulated that castration had prevented completion of immunologic maturity. In sheep, which are ruminants closely related to goats, evidence was presented by Schinkel and Ferguson[11] that immunologic maturity is completed before birth. Fetal lambs (which probably were agammaglobulinemic!) rejected, within 18 days, skin homografts *in utero*. In view of this evidence it would have been surprising if the function of the homotransplanted kidney had been prolonged in castrated goats.

Professor Gustave Dammin of the Peter Bent Brigham Hospital, Boston, very kindly examined samples of the histologic preparations and expressed the opinion that the histopathology was unique, as compared to that in man and the dog. The abnormal changes in the endothelial and muscular layers of the small arteries and arterioles, and the atrophy of the tubules were strikingly less far advanced.[1]

To increase further the knowledge about transplantation in general, and specifically about the possible causal relationships between the relative lack of endothelial changes, the better preservation of the tubules, and the longer survival times, additional studies with homotransplanted kidneys in goats are planned.

SUMMARY

Kidney homotransplants in castrated and noncastrated male American Angora goats secreted urine for 17.8 ± 6.9 days, the longest period reported for any animal except man. The results were essentially the same, in every respect including total time of urine secretion and histopathology, for the four donor-host combination groups studied, *viz.*, castrated males to castrated males, normal bucks to normal bucks, castrated males to normal bucks, and normal bucks to castrated males. The histopathology was somewhat unique, however; the inflammatory changes in the arteries and the atrophic changes in the tubules were less advanced in these homotransplanted kidneys in goats than in those reported by others in dogs and man.

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JEROME COCHRAN: THE GUIDING GENIUS IN PUBLIC HEALTH LEGISLATION IN ALABAMA

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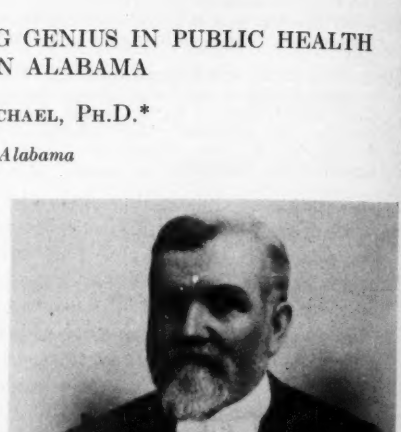
Dr. Jerome Cochran, author of much of that which appears in the Statutes of Alabama in relation to public health and medical practice, opened his office in Mobile in 1865.

The Cochran family has become widely disseminated in America since two brothers settled in this country; one in Virginia and one in Pennsylvania, many years before the Revolutionary War. The Virginia branch of the family increased rapidly and after spreading over many counties of the Old Dominion overflowed into Georgia, Tennessee, Alabama, Mississippi and Texas.

Jerome Cochran was born in Moscow, Fayette County, Tennessee on December 4, 1831, while that country was sparsely settled with white citizens, and largely occupied by Choctaw and Chickasaw Indians. He was the second child and first son of Augustine Owen Cochran and Frances Baily. While Jerome was a small child, his family moved to Marshall County, Mississippi, where his father spent the greater part of his time as a cotton planter. Young Jerome worked in the fields along with his father's slaves and received very little formal education except during the summers at an old field-school, after the farm work was finished. Later on, he supplemented his poor beginning by an extensive course of reading and private study which covered mathematics, logic, philosophy, poetry, general science, literature and modern languages.

When he was 19, he became a country school teacher, a position which he held for the next 6 years. This gave Jerome time to continue his reading and study in various fields. In 1855, he married Miss Sarah Jane, daughter of Jared Collins, a well-to-do farmer of DeSota County, Mississippi. The same year, he met Dr. Robert H. Harrison, Professor of Materia Medica and Therapeutics at the Botanic Medical College, Memphis, who induced Jerome to enter the study of Medicine. After two courses of lectures, he graduated as Doctor of Medicine in 1857. Although he entered the school without preliminary preparation, he was soon at the head of his class and delivered the valedictory address. While a student at Memphis, Jerome became conscious that the botanic system of instruction was untenable in certain areas. So after 2 years of practice in North Mississippi, he entered the medical department of the University of Nashville, in the fall of 1859. In February 1861, along with 153 others, Dr. Jerome Cochran received the M.D. degree, his second one and again he was the one selected to deliver the valedictory address.



On April 12, 1861, the first shot of the Civil War was fired at Fort Sumter. Soon after the war broke out, Dr. Cochran spent some time with the troops at Corinth, Mississippi and later on was

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associated with the 3000-bed Confederate Hospital at Okalona, Mississippi, the largest confederate hospital outside of Richmond. Dr. Cochran received the rank of Surgeon in the army early in 1862 and remained on duty at Okalona until the post was abandoned after the battle of Corinth.

The remanents of the hospital were transferred to Meridian, Mississippi, and to Marion Station, a few miles north of that city. Dr. Cochran remained at Marion Station until General Sherman advanced on Meridian after the fall of Vicksburg in 1864. He loaded most of the hospital equipment on railroad cars and shipped all of it to Mobile. Then he was ordered to open a hospital in Tuscaloosa and just as it was ready for occupancy, he was ordered to turn it over to Dr. Anderson and proceed to Gainesville in the capacity of surgeon of the post. The order disturbed Dr. Cochran so much that he wrote a severe letter to Dr. Preston B. Scott, acting Medical Director, who detailed him for examination of conscripts for several North Alabama counties. He had headquarters at Tuscaloosa and was on that assignment when the Civil War ended.

During the next few months he turned his attention to the study of mental diseases under the guidance of Dr. Peter Bryce, the Superintendent at the State Insane Hospital at Tuscaloosa.

One day late in June 1865, this Confederate Army veteran, a perfect stranger without friends in Alabama, moved to Mobile and nailed up a sign near his quarters which read, Jerome Cochran, M.D. From that humble start, his practice grew rapidly and he soon was ranked with the most successful practitioners in Alabama.

When the Medical College of Alabama was opened after being closed for 7 years because of the war, Dr. Cochran was elected Professor of Chemistry. In 1875, he was appointed Professor of Public Hygiene and Medical Jurisprudence, a position which he held for 2 years.

Dr. Cochran's connection with the Medical Association of Alabama began in 1868. Pursuant to a call of the Selma Medical Society, delegates from that society, the Greensboro Medical Society, the Montgomery Medical and Surgical Society, and the Mobile Medical Society, together with other medical gentlemen of the State, met in the city of Selma, in the Hall of the Chamber of Commerce at 3:00 p.m., March 3, 1868, for the purpose of reorganizing the Alabama State Medical Association after 7 years of suspension due to the consequences of the war. At this meeting, he was elected Recording Secretary, an office which he held for the next 6 years. He also served as chairman of the Publishing Committee from 1868 to 1872. The Association, like most other medical associations in America, was a simple convention of doctors who were loosely bound together but with very few duties and no penalties. In the discharge of his duties as secretary, Dr. Cochran soon discovered the inherent defects and weaknesses of this type of organization or association. His avowed aim from the beginning was to replace this loose convention of doctors by a compactly organized body which would be thoroughly disciplined but a self-perpetuating medical legislature. All of this is clearly expressed in the second section of his famous constitution:

"The objects of this Association shall be to organize the Medical Profession of the State in the most efficient manner possible; to encourage a high standard of medical education, and regulate the qualifications of practitioners of medicine within the state; to promote professional brotherhood and encourage a high standard of professional ethics; to combine the influence of all the medical men in the State so as to secure by legislative enactments their own legitimate rights and privileges, and the protection of the people against all medical quackery and humbug; to encourage the study of the medical botany, medical climatology, and medical topography of the State; to secure careful and reliable accounts of all the endemic and epidemic diseases of the State; in a word, to watch over and protect, encourage and aggrandize all the interests of the Medical Profession of the State."

Dr. Cochran presented his plan to the association at the annual session in 1870 at Montgomery. His plan took the shape of a series of resolutions and the special committee for considering the proposal rendered an adverse report on most of Dr. Cochran's suggestions. The plan was formulized as a new constitution and presented at the next meeting in 1871 at Mobile. The plan of the constitution was discussed and referred to a committee of five, to report at the next annual session at Huntsville in 1872. After considerable discussion at this meeting, Dr. George A. Ketchum, Professor of Medicine, Medical College of Alabama, moved that action on the new consti-

tution be postponed until the morning of the second day of the next annual meeting. It came up for final action at the Tuscaloosa meeting in 1873 and was adopted.

Two of the special features of the Constitution were: (1) a House of Counsellors restricted to 100 members who would control the policy of the association and (2) a Board of Censors, composed of 5 counsellors to pass on the business of the association. The Constitution was supplemented by a code of ordinances which appear in "The Book of the Rules." These ordinances were also framed by Dr. Cochran.

Dr. Cochran's interest in the field of public hygiene was evidenced by a series of articles, which appeared in the Mobile Register in 1870, on "The Origin and Prevention of the Endemic and Epidemic Diseases of Mobile." These articles attracted so much attention that the City of Mobile adopted a health ordinance which was authored by Dr. Cochran. This ordinance created a health officer and a Board of Health to be elected by the Mobile Medical Society. The Board of Health was in charge of sanitary supervision in the city. Dr. Cochran was elected the first Health Officer and served for 2 years until the Republicans came into power and abolished the Board of Health. Following a yellow fever epidemic in 1874, Dr. Cochran was reelected Health Officer and this time his plans of action were adopted. Within a short time, he instituted a program of vaccination, quarantine, disinfection and isolation. The epidemic, which was city wide, was pressed back step by step until it was utterly exterminated.

The American Medical Association, at its annual session in 1871, appointed a committee composed of one member from each state to urge upon the legislatures of the several states the creation of State Boards of Health. There were two states, California and Massachusetts, with State Boards of Health at that time. Both were organized on the same plan with seven members in each state. The members were appointed by the Governor from time to time and were selected partly from the medical and partly from other professions. The committee which was appointed by the American Medical Association adopted the above plan. The plan was presented to the General Assembly of Alabama in the winter of 1871–1872 by Dr. J. S. Weatherly, Alabama member of the Committee on the American Medical Association. Dr. Cochran did not approve this plan and set about to defeat it, which he did. He then submitted his own plan at the next State Association meeting in Huntsville in 1872 and the other plan was also submitted.

In discussing the two plans, Dr. Cochran remarked as follows:

"I am free to say that I have no special objections to the Massachusetts and California plan, in itself. If no other arrangement was suggested, I would very cheerfully advocate it, and do all I could to secure its adoption by the Legislative authorities of the State. A State Board, so constituted, would undoubtedly render valuable service to the State, and make valuable contributions to our knowledge of the natural history of disease and of the influence of various circumstances, geological, topographical and meteorological, upon the sanitary conditions of localities. If, then, I oppose this system of organizing a health department for the State, it is not because I think it intrinsically bad, but simply because I think I have a better plan to offer to your consideration. . . .

"I would have a general health law passed by the Legislature of the State, carefully prepared, so as not to stand in need of frequent revision or amendment.

"I would have this law to invest the Medical Association of the State with the functions, powers and responsibilities of a State Board of Health; and these functions I would have exercised through such organs as the Association, which in its wisdom, might think best.

"I would have the same act of the Legislature to invest each county medical society with the functions of a county board of health, said functions to be exercised through such organs as the said county societies should, in their wisdom, determine to be best."

After thorough discussion, Dr. Cochran's plan was endorsed by a majority at the Huntsville meeting in 1872. It was endorsed again by the unanimous vote of the Association at the annual session in Tuscaloosa in 1873. His plan was in the form of a bill embodying details and the Board of Censors were ordered to present it to the next General Assembly. It was enacted into a law of the State on February 19, 1875. This law makes the State Medical Association, the State Board of Health and the County Medical Societies in affiliation with it, the County Boards of Health.

In order to facilitate the action of the Association in reference to this matter, the Board of Censors which was chairmaned by Dr. Cochran, presented the following resolutions which were recommended and adopted:

"*Resolved*, That the Medical Association of the State of Alabama hereby accept for themselves and for the County Medical Societies under their jurisdiction the provisions of an act entitled an Act to establish Boards of Health in the State of Alabama, and approved by the Governor on the 19th day of February, A.D. 1875; and will endeavor to discharge the duties assigned to them in said act in good and with earnest purpose, to be of service to the people of Alabama.

"*Resolved further*, That a copy of these resolutions, properly certified by the President and the Secretary of the Association, be transmitted to the Governor of the State."

The State Association's annual orator in 1878 referring to Dr. Cochran as the genius of medical organization had this to say in his presence.

"Our medical association, . . . owes its present excellence and preeminence largely to the zeal, fidelity and energy of one mind. Patient, far reaching, tenacious, learned, indefatigable, oftentimes misunderstood, and sometimes misrepresented, Dr. Jerome Cochran builded wiser than he knew in creating the plan of the State Association. He deserves to rank as the apostle of organized medical action in the new era."

Although the chief planning for the organization of the Alabama Medical Association was crystallized by Dr. Cochran in the early seventies, the association served both as an inspiration and a model for other state medical associations many years later. No doubt the recognition of his labors, by distinguished practitioners, was a great source of satisfaction to him.

Dr. Charles A. L. Reed, in "The President's Address" at the American Medical Association's annual meeting at St. Paul in 1901, made the following statement about the Alabama Association: "Alabama always progressive, but the happy possessor of other conditions, was able to place the regulation of medical practice for the time being under the control of its incomparable State Medical Association."

Dr. E. L. Marechal in his presidential message in 1902 in Birmingham referred to the organization of the Association as follows:

"Careful and intelligent investigation of the system of organization which this Association has adopted would obviate much of the misapprehension that exists relative to its purposes. Justice and fairness alike demand that criticism should be based on knowledge. This Association has received unstinted commendation from those who have given thought and study to it. . . . Dr. George H. Simmons, Secretary of the American Medical Association, and editor of its *Journal*, in some remarks which be made during a recent visitation upon a meeting of the Mobile County Medical Society, characterized this Association as 'the best medical organization in the world.' The American Medical Association in its plan of reorganization, adopted the fundamental principles underlying our system. That Association is a federation of State societies. This Association is a federation of county societies. From all over this vast and progressive country of ours inquiries are being made as to the specific plans and purposes of this Association. The evident and frequently expressed purpose of such inquiries is to give our plan careful study, with a view of engrafting its principles into the organic law of other State organizations."

Dr. Cochran not only was an active member of the State Medical Association but he played many important roles in the American Medical Association. He served as an official delegate to the American Medical Association from both the Mobile Medical Society and the State Medical Society. He was a member of the nominating committee of the American Medical Association at several annual meetings and served as chairman of that committee in 1894. He was a member of the committee on State Medicine for many years and also served as chairman of the committee on "Medical Jurisprudence—Chemistry and Psychology" in 1874.

Probably the greatest demonstration of confidence in Dr. Cochran's ability as a medical leader was when, in 1894, he was appointed Chairman of the Special Committee of the American Medical Association to petition Congress to create a Department and Secretary of Public Health. The committee was authorized at the 1891 annual meeting and he had been a member of the committee since the 1893 meeting. Following his report of the committee at the Atlanta meeting, May 1896, he was authorized to appoint as many members as might be needed to achieve their assignment. A letter to the Editor

of the *Journal of the American Medical Association* dated July 6, 1896, indicated that he had been sick for some weeks but that he had made some progress toward the proposed enlargement of the committee. His death occurred before the next annual meeting of the American Medical Association and the efforts of the committee seem to have been collapsed soon thereafter. No doubt the accomplishments of the committee would have been quite different had Dr. Cochran lived a few more years. In any case, the first Secretary of Health, Education and Welfare, Mrs. Oveta Culp Hobby, was appointed by President Eisenhower 57 years later, in 1953.

Dr. Cochran's literary record is quite extensive. His scientific papers usually appeared in the annual volumes of the *Transactions of The Medical Association of the State of Alabama*. The titles of his papers cover a wide range of subjects as indicated by a random sampling which follows: "Administration of Chloroform by Deglutition"; "Endemic and Epidemic Diseases"; "Yellow Fever"; "Hemorrhagic Malarial Fever"; "The White Blood Corpuscle, its Physiology and Pathology"; "Smallpox"; "Puerperal Fever"; and "Sanitary Administration"; "The Theory and Practice of Quarantine"; and "The Value of Vital and Mortuary Statistics."

One of his biographers, in 1880, made the following statements concerning his characteristics. "Intellectually he is of the most imposing proportions; and there is no exaggeration in saying that he is one of the most learned men of the South. . . . A fine command of language, a chaste and luminous style, aptness for philosophical speculation, large powers of analysis, comparison and reflection, a self-control which is never disturbed, and a self-reliance based upon a consciousness of strength, give him absolute command of his resources and render him at once an able writer, an instructive and interesting talker, and a consummate master of debate. . . . He has been known to persevere in his convictions in regard to an important public professional interest and to struggle for their ascendancy; notwithstanding, it involved the alienation of friends and the sacrifice of private interests. . . . Although not insensible to praise, and fond of a display of his talents, he cannot be considered vain. . . . A large head, a thoughtful and somewhat saddened brow, a firm mouth, a quiet dark grey eye, and a complexion rendered sallow by ill-health and sedentary habits, give ordinarily an air of stoical apathy to his well-formed, intellectual, and rather attractive face."

Dr. Cochran died on August 17, 1896, at Montgomery after serving for almost 20 years as the first State Health Officer. Dr. and Mrs. Cochran had two living children at the time of his death, Edmund Collins Cochran and Jerome Bowling Cochran, both businessmen in Houston, Texas.

The expression of the State Board of Censors at the next annual meeting was appropriately brief: "During the last year a great calamity befell us in the death of our leader (Jerome Cochran), the man who conceived this organization, and, above all others, built it up and made it what it is. Let us hope that his work was so well done that, although he is no longer here to guide us with his wisdom and to animate us with his courage, we may be able to push on and reap that splendid fruition for which he so earnestly worked and eagerly longed."

In 1898, the Alabama State Association President, Luther L. Hill, recommended establishing the "Jerome Cochran Lecture" to be given at each future annual meeting of the Association. The first lecture was delivered the next year by Dr. J. T. Searcy of Tuscaloosa. The list of Jerome Cochran Lecturers includes medical men not only from Alabama but also some of the most renowned physicians this country has produced.

At the time of his death, a biographer summarized Dr. Cochran's professional activities and contributions which appeared in the *Journal of the American Medical Association*: "Dr. Cochran was one of the most active health officers in the United States, and a man of phenomenal energy. His sanitary work at Decatur, Alabama, in 1888, was intelligent and thorough. As a man, his character was that of one devoted to sanitary science, of great learning and unwearied industry. His long and faithful service to his State is such that it cannot be overrated or excluded from the history of its progress. He had his 'quips and oddities,' what genius has not? But take him all in all, we shall not see his like. His friends, his State, his country and our Association have suffered a great loss."

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SQUAMOUS CELL CARCINOMA OF THE FLOOR OF THE MOUTH, ALVEOLAR RIDGE, AND BUCCAL MUCOSA

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Although cancer of the oral cavity is generally reported to occur about three times more frequently in men than in women, 9 of the 12 patients seen at the Sheffield Tumor Clinic have been women. This reversal of the national ratio is thought to be due to the prevalence of the use of snuff among women in this region. In a rather extensive study, Wynder and associates[10] concluded that tobacco is an important factor in the development of cancer of the oral cavity. Poor oral hygiene, ill fitting dentures and other forms of chronic irritation do not appear to play a significant role in the etiology according to this report.

Martin and Koop[6] have stated that the mucous membrane changes of avitaminosis B are more common and of more importance than those of all the other chronic irritants. Certainly one must agree that in the average case more than one irritant is demonstrable. The patients studied here have all used tobacco in some form, but it cannot be denied that they have all had poor oral hygiene and evidences of poorly balanced diets. They have all had leukoplakia of the mouth.

Theoretically, all of these lesions must pass through a curable stage, but if patients are to be prevented from dying of the disease, a form of adequate therapy must be decided upon, and instituted before they become incurable. It is often stated that many of these patients wait too long before seeking medical attention, and lack of education of the public in this respect is blamed. To some extent this may be true, but too often the failure to obtain a cure is due to inadequate treatment by the physician.

TREATMENT

Two forms of accepted treatment are radiotherapy and surgical extirpation. Various combinations of these have been used. Clifton and Hardin[2] believe that the best results can be obtained from irradiation followed by radical surgery. They advocate giving a full course of radiotherapy and if there is any suggestion of residual disease, surgery is then instituted. Fletcher[3] advocates interstitial radium therapy alone or combined with external irradiation for early uncomplicated lesions of the buccal mucosa. If bone is involved, supervoltage roentgenotherapy is given and a few weeks later a "commando" procedure is used. In the floor of the mouth or lower gum he uses various combinations, individualizing the therapy. Lane[4] has stated that if a combination of radiotherapy and surgery is to be used it is preferable to do surgery after the irradiation because surgery interferes with the blood supply to such an extent that adequate radiotherapy cannot be administered. He also states that the principles involved in the argument for preoperative irradiation are: (1) prevention of spread of tumor, (2) lowered possibility of seeding and (3) conversion of an inoperable lesion to one that is accessible to surgery. Conversely, wound healing is poor in previously irradiated tissues and much postoperative morbidity is encountered in this respect. When a lesion has been diminished in size by irradiation, it is difficult to be sure that the margins of excision are adequate because residual tumor may be present in areas that appear free of it.

A convincing argument against preoperative irradiation is that valuable time is lost while this treatment is being carried out and while waiting for the tissue reaction to radiotherapy to subside. Mattick and associates[7] reported that in a series of 82 patients treated by radiotherapy alone there were 15 cures; 6 of these, however, had serious complications from the therapy. One must agree with them, that based on these figures the renewed surgical attack on intraoral cancer seems justified. This tends to agree with the findings of Slaughter and associates,[8] that it is practically impossible to sterilize squamous cell carcinoma in bone by irradiation, and in the occasional case where this is accomplished the jaw will be destroyed by the therapy.

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necessitating its removal for radionecrosis and osteomyelitis.

Early involvement of the mandible must account for many of the failures to obtain a cure with radiotherapy. Ward and Robben[9] cite the establishment by various authorities that in 50 per cent of normal individuals the lymphatics of the tongue and floor of the mouth pass through the periosteum of the mandible on their way to lymph nodes in the submaxillary triangle. In their opinion, this accounts for the early attachment of metastases and primary growth to the mandible and emphasizes the need for removal of the periosteum or of the entire horizontal ramus, when the primary growth extends close to the lower jaw. Because the mandible will be involved microscopically for some time before this involvement can be detected radiographically or clinically, it appears that earlier resection of the mandible is essential if the cure rate is to be increased; regional lymph node metastases must also be present in many of these patients prior to the development of palpable lymph nodes. It seems as logical to do a suprahyoid neck dissection routinely, in these cases, as it is to do an axillary dissection for carcinoma of the breast.

The suprahyoid neck dissection seems to be adequate at the present time if palpable lymph nodes are absent; however, in time a complete unilateral neck dissection may prove to produce superior results.

If lower cervical metastasis is suspected either upon initial examination or following excision of the primary growth and suprahyoid dissection, it is expedient that a complete unilateral neck dissection should follow.

The amount of mandible that should be removed is often difficult to determine. Slaughter and associates[8] state that if bone is invaded, or if the lesion lies posteriorly in the floor of the mouth, gingiva or buccal mucosa, the entire hemimandible should be removed. Lesions lying well anterior with no bone invasion require removal of the horizontal ramus. A much better cosmetic result is obtained if more than one-half of the mental process can be saved. This can be done with relative safety if the lesion lies posteriorly and especially if no bone invasion is detectable.

Plastic reconstruction can be carried out with much greater success when the ascending ramus can be left intact. Castigliano[1] has reported the successful use of a Vitallium prosthetic replacement for an entire hemimandible at the time of resection after radiotherapy. However, he points out that few cases of malignancy involving the mandible are suitable for this type of prosthesis. The major objections to bone grafts or prosthetic replacements at the time of hemimandibulectomy are that these tend to interfere with healing and also obscure recurrences of the carcinoma.

The deformity that results from hemimandibulectomy, with or without radical neck dissection is not nearly so severe as would be expected by one unfamiliar with it. The patients are usually in the older age group and not overly concerned with their appearance. In fact very few of them will consent to later plastic repair of their defects. The functional result is satisfactory and even if they are not cured of their disease, most of them are made more comfortable by the elimination of the primary neoplasm from their mouths.

The 12 patients that have been treated in the Sheffield Tumor Clinic will be discussed briefly in the following paragraphs. The experiences related here should be of some value to one who deals with this disease.

CASE REPORTS

Case 1. The patient, a white woman, aged 54, was presented to the clinic July 3, 1953, with a squamous cell carcinoma of the left buccal mucosa, extending anteriorly from the position of the second molar tooth, 1.5 cm. in diameter, and reaching the gingiva. Leukoplakia was present around the area of the lesion which had been present about 4 months. On July 22, 1953, the lesion was locally excised and the defect was allowed to granulate. There was prompt local recurrence on the alveolar ridge and on September 29, 1953, a second local excision was done. Local recurrence on the alveolar ridge was recognized 1 month later and on February 10, 1954, the lesion was locally excised the third time. Again the carcinoma recurred locally. There was still no radiographic evidence of bone invasion and no enlarged nodes were palpable in the neck. On May 3, 1954, a partial mandibulectomy was done, removing a portion of the floor of the mouth and buccal mucosa. The anterior portion of the mandible was preserved and the remainder of the left hemimandible was removed. No actual suprahyoid dissection was done. No tracheotomy was performed and the patient took oral feedings on the 3rd postoperative day. Healing was prompt; a good cos-

metic result was obtained and there has been no recurrence of the disease.

Case 2. The patient, a white woman, aged 76, was presented to the clinic on July 15, 1955. A squamous cell carcinoma of the right buccal mucosa had been diagnosed elsewhere in February 1952, at which time the patient reportedly had cervical adenopathy on the right. She was given radiotherapy. Radionecrosis of the mandible developed and she was operated on in May 1954, and again in October 1954, for removal of sequestra. A large fistula then developed through the right cheek. When first seen in this clinic the fistula was present and most of the posterior mandible was absent. Around the margins of the fistula, recurrent tumor was present along with severe leukoplakia. She could not chew and could barely open her mouth enough to take liquids. Surgical extirpation of the lesion was advised but she refused further treatment and died of the disease on February 9, 1956.

Case 3. The patient, a Negro man, aged 83, was presented to the clinic on July 29, 1955, with a squamous cell carcinoma, 1.5 cm. in diameter, on the posterior right lower alveolar ridge extending slightly into the floor of the mouth. This had been present about 6 weeks. There were enlarged lymph nodes present in the right submaxillary region. There was no radiographic evidence of bone invasion. Leukoplakia was present in patches throughout the mouth. On August 9, 1955, a resection of the floor of the mouth and right horizontal ramus of the mandible, combined with a suprahyoid neck dissection, was done. None of the lymph nodes removed were positive for carcinoma. Tracheotomy was performed at the close of the procedure and tube feedings were given for 4 days. A slight infection developed in the mucosal wound but this cleared completely. A small area of recurrent cancer was found in the buccal mucosa, 4 months later, which was locally excised and there has been no further evidence of recurrence.

Case 4. The patient, a white woman, aged 55, was presented to the clinic on November 11, 1955, with a squamous cell carcinoma, 2.0 cm. in diameter, on the lower gingiva in the midline, extending onto the buccal mucosa and onto the floor of the mouth. This had been noticed first in January 1953, and had been locally excised elsewhere in July 1953. Following excision it promptly recurred. Leukoplakia of the mouth was present and there was radiographic evidence of involvement of the mandible on both sides of the symphysis. No cervical adenopathy was found. On November 22, 1955, an operation was performed, resecting the anterior portion of the floor of the mouth and central portion of the lower lip and buccal mucosa along with the anterior one-half of the right and left mandible. An attempt was made to reconstruct the chin using a curved Steinmann pin. Tracheotomy was not done. Tube feedings were given. By the 3rd postoperative day the mucosa had sloughed over the prosthesis and the wound was infected. Two weeks later the prosthesis was removed and many weeks later the wound healed. In May 1956 a recurrence of cancer was found on the remaining portion of the left horizontal ramus which was locally excised. This recurred within 1 month and further surgery was advised. The patient refused further therapy until October 1956. At that time, there was extensive spread of carcinoma onto the floor of the mouth and buccal mucosa. On September 5, 1956, a wide resection of these areas along with the remainder of the left mandible, except for a small portion of the ascending ramus, combined with a left suprahyoid neck dissection was done. Tracheotomy was performed and tube feedings were given. Healing was prompt; the patient got along well for about 4 months and then developed enlarged lymph nodes in the right side of the neck. On May 10, 1957, a right radical neck dissection was carried out and the nodes found there were positive for metastasis. The patient got along well after this procedure and there has been no evidence of further recurrence.

Case 5. The patient, a white woman, aged 51, was presented to the clinic on May 4, 1956. In November 1955 she had been diagnosed elsewhere as having squamous cell carcinoma of the left floor of the mouth posteriorly with enlarged lymph nodes in the left submaxillary region. She was given radiotherapy and the lesion in the mouth reportedly healed temporarily. When seen here, there was an ulcerating lesion in the left floor of the mouth, 3.0 cm. in diameter, extending across the posterior alveolar ridge onto the buccal mucosa. There was an 8- by 6-cm. mass extending from the floor of the mouth around the angle of the mandible inferiorly. There were no enlarged lymph nodes palpable elsewhere in the neck. There was no radiographic evidence of bone involvement. There was extensive leukoplakia of the mouth. On June 27, 1956, the lesion in the floor of the mouth was excised and the mass was resected off the mandible. There was a great deal of inflammatory reaction in the area and a large amount of caseous material was encountered in the region of the mass, arousing so much suspicion of tuberculosis that the operation was terminated at this point. The left submaxillary gland and some adjacent lymph nodes had been removed; subsequent study revealed squamous cell carcinoma in these. No tuberculosis was found. A wound infection and fistula developed along with recurrent carcinoma in the floor of the mouth. On October 15, 1956, a radical neck dissection was

done along with left hemimandibulectomy and resection of the floor of the mouth. Widespread cervical lymph node metastases were found. Again, wound infection and a persistent fistula developed along with recurrent carcinoma in the mouth extending outward along the fistulous tract. The patient died of this disease on January 2, 1957.

Case 6. The patient, a white woman, aged 66, was presented to the clinic on December 14, 1956. She gave a history of having had a lesion on the lower gingiva for 4 years. When examined she was found to have squamous cell carcinoma extending onto the floor of the mouth to the anterior tonsillar pillar and across the alveolar ridge with extensive involvement of the buccal mucosa almost up to the superior gingiva. There was extensive leukoplakia of the mouth. Radiographically, there was involvement of the horizontal ramus of the mandible from the cuspid area to the angle. No cervical adenopathy was detected. On December 21, 1956, a left radical neck dissection was done *en bloc* with a wide resection of the floor of the mouth and buccal mucosa. The mandible was removed from a point about 1 cm. beyond the symphysis to the mid-portion of the ascending ramus. No cervical lymph node metastasis was found. Tracheotomy was performed and tube feeding was carried out. Healing was prompt and there has been no evidence of recurrence.

Case 7. The patient, a white woman, aged 52, was presented to the clinic on March 22, 1957. She had squamous cell carcinoma, 2.5 cm. in diameter, in the mid-portion of the floor of the mouth on the left, extending from the base of the tongue almost to the lower gingiva. This was surrounded by extensive leukoplakia. The lesion had been present about 4 months. No cervical adenopathy was detected. On March 3, 1957, a left radical neck dissection was done combined with a wide resection of the left floor of the mouth, including a small portion of the base of the tongue. The left mandible was removed *en bloc* from 1 cm. beyond the midline anteriorly to the angle. Tracheotomy was performed and tube feeding was carried out. No metastasis to bone or lymph nodes was found. Primary healing occurred and there has been no evidence of recurrence.

Case 8. The patient, a Negro woman, aged 80, was presented to the clinic on July 12, 1957. She gave a history of having had a sore in her mouth for 3 months. She was found to have squamous cell carcinoma involving almost the entire lower alveolar ridge on the right with extensive involvement of the buccal mucosa. There were many enlarged lymph nodes palpable in the right side of the neck. There was extensive leukoplakia throughout the oral cavity. The patient's general physical condition was very poor; it was believed that she could not tolerate radical surgery, and radiotherapy was given. The patient is still living but has been unable to return to the clinic for several months. Her present condition is unknown, but the disease is still present and progressing.

Case 9. The patient, a white man, aged 63, was presented to the clinic on January 24, 1958. He gave a history of having had a sore in his mouth for 10 weeks. He was found to have a squamous cell carcinoma, 5.0 cm. in diameter, extending over the entire right lower alveolar ridge and across the floor of the mouth almost to the border of the tongue and laterally onto the buccal mucosa. There was extensive leukoplakia of the mouth and lips. There were enlarged lymph nodes palpable in the right supraclavicular region. There was radiographic evidence of involvement of the right mandible from the cuspid area to the molar area. On February 6, 1958, a right radical neck dissection was done *en bloc* with a wide resection of the right floor of the mouth and buccal mucosa. The right mandible was removed from 1 cm. beyond the symphysis to the upper portion of the ascending ramus. A small portion of the right side of the tongue was removed with the floor of the mouth. Tracheotomy was performed and tube feeding was carried out. No cervical lymph node metastasis was found. Healing occurred promptly and there has been no evidence of recurrence.

Case 10. The patient, a white woman, aged 75, was presented to the clinic on April 18, 1958. She had developed a sore on the left buccal mucosa in December 1957 and was treated elsewhere with radiotherapy without biopsy. This sore reportedly healed, but soon she developed another sore on the left alveolar ridge, posteriorly. When seen here she had an ulceration, 2.0 cm. in diameter, over the left inferior alveolar ridge, posteriorly, which extended onto the buccal mucosa. This was surrounded by leukoplakia. There were some enlarged lymph nodes in the right submaxillary region. The lesion was biopsied and it proved to be squamous cell carcinoma. There was no radiographic evidence of metastasis to the mandible. On May 16, 1958, a left radical neck dissection was done combined with resection of the mandible and floor of the mouth and buccal mucosa around the lesion. The left mandible was removed from 1 cm. to the left of the midline anterior to the ascending ramus. Tracheotomy was performed and tube feeding was carried out. No cervical lymph node metastasis was found. There was an infection of the wound inside the mouth but this cleared after several days. The healing process was slow. There has been no evidence of recurrent carcinoma.

Case 11. The patient, a white man, aged 66,

was presented to the clinic on September 19, 1958. After having two molar teeth extracted, in January 1958, he developed a sore in that area on the left inferior alveolar ridge. His dentist referred him to a physician who proved this to be squamous cell carcinoma by biopsy. He was given radiotherapy. The lesion continued to grow and, when seen here, had extended along the entire lower alveolar ridge almost to the midline, anteriorly, and onto the floor of the mouth and buccal mucosa. In addition, a fistula had formed through the left cheek and the carcinoma was growing out through this to the skin margin. There was extensive leukoplakia of the mouth and lips. No cervical adenopathy was detected. Radiographically, the entire left hemimandible appeared to be involved with carcinoma. On October 1, 1958, a left hemimandibulectomy was done with resection of the floor of the mouth and buccal mucosa and a suprahyoid neck dissection *en bloc*. Anteriorly, the mandible was resected 1 cm. beyond the symphysis. Tracheotomy was performed and tube feeding was carried out. Lymph node metastasis was found in the submaxillary region. Primary healing occurred and there has been no evidence of recurrence.

Case 12. The patient, a white woman, aged 70, was presented to the clinic on January 17, 1958. She gave a history of having had a sore in the mouth for several months. When examined she was found to have squamous cell carcinoma of the right buccal mucosa, extending from the ascending ramus of the mandible to the corner of the mouth and from the soft palate to the floor of the mouth around the anterior pillar of the tonsil. There was extensive leukoplakia of the mouth. No lymph node enlargement was found and radiographically the mandible was not involved. The patient was in very poor condition physically and did not wish to have surgical treatment. Radiotherapy was given; the intraoral lesion was seen to advance in spite of this and cervical adenopathy developed. In May 1958 she was found to have subcutaneous nodules on her back and upper and lower extremities. These proved to be metastatic squamous cell carcinoma when biopsies were taken. The patient died of the disease on June 10, 1958.

From studying these cases it seems fairly clear that radiotherapy cannot be depended upon to cure squamous cell carcinoma of the oral cavity and it seems questionable that it can be depended upon for any degree of palliation. It seems obvious that local excision of lesions in the floor of the mouth, alveolar ridge and buccal mucosa is rather futile and apt to lead to disastrous delay in starting adequate treatment. Preoperative radiotherapy in the cases seen here has contributed only to increased difficulties in managing the cases; however, none of these patients had planned radiotherapy specifically in preparation for surgery so that this does not represent a fair appraisal of this approach.

None of the patients in this series has a 5-year cure yet, but most of the recurrences seem to appear within a few weeks or months. When a year has gone by without recurrence, it is thought that the patient has a good chance of being cured, although a few will get recurrence after a much longer period of time.

SUMMARY

The cause of squamous cell carcinoma remains unknown. In the oral cavity its origin seems to be associated with chronic irritation. Recent studies point to the use of tobacco as one of the most significant irritants. Leukoplakia appears to be a forerunner of the development of squamous cell carcinoma in a high percentage of cases.

Patients are seeking treatment for carcinoma of the oral cavity much too late, and more work needs to be done in the field of public education. If the public could be made aware of the possible significance of leukoplakia, much would be accomplished.

In treating squamous cell carcinoma of the alveolar ridge, floor of the mouth and buccal mucosa, early radical surgery appears to give the best results. The experience here seems to indicate that better results are obtained without preoperative radiotherapy. It now appears that the ordinary case without any evidence of cervical metastasis should have a wide resection of the lesion, including a variable amount of the mandible, and a suprahyoid neck dissection. The amount of mandible removed depends upon the location of the lesion and the degree of involvement of bone judged to be present. It appears that adequate resection of the mandible is extremely important and the cosmetic result should always be considered secondary to obtaining a cure. Martin[5] has stated that the permissible limits of radical surgery of the head and neck are determined only by the question of necessity for removal of essential structures without which patients cannot survive. When cervical lymph node metastasis is suspected, radical neck dissection should be done and this should be done bilaterally if necessary.

It becomes increasingly obvious that the only way to obtain a better rate of cure is through more adequate surgical treatment in the earlier stages of the disease. There is no satisfactory method of palliation in this disease. At any age and in almost any condition, the slightest chance of cure is worth almost any risk.

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SURGICAL MANAGEMENT IN COMPLICATIONS OF DIVERTICULITIS OF COLON*

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The concept in the treatment of recurrent, severe diverticulitis has changed during the past decade. Before that time, treatment consisted primarily in watchful waiting and hoping that the patient would improve and that no complications would develop. This often led to disastrous results.

Since diverticulitis is primarily a disease of the older age groups, and the average age of our population is so rapidly increasing, we may expect to encounter diverticulitis and its complications more frequently than in the past. Many of these complications require extensive and, sometimes, heroic measures for correction. In order to avoid these serious and often fatal complications, a more positive approach in the treatment of recurrent diverticulitis is recommended.

Notwithstanding the recommendations for preventive treatment, we, in our hospital, continue to receive numerous patients with serious complications of diverticulitis. This paper concerns the treatment of these complications.

REVIEW OF LITERATURE

The clinical significance of diverticulitis first became appreciated at the beginning of this century through the writings of Fischer,[4] Beer,[1] Moynihan[9] and Mayo and associates.[8] In 1904 Beer[1] reported 18 cases of diverticulitis and presented the first significant treatise appearing in American literature. Beer's description of the disease leaves little to be added, even at the present time. His 18 cases contained most of the complications of diverticulitis, such as partial obstruction, perforation with localized abscess, perforation with generalized peritonitis and sigmoidovesicle fistula. His group did not include the complications of hemorrhage, sigmoidocutaneous fistula and sigmoidoenteric fistula, which have subsequently been reported.

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The incidence of diverticulitis in the population at large is unknown. Diverticulosis occurs with equal frequency in men and women, while diverticulitis, requiring surgical treatment, occurs more often in men. In patients upon whom roentgenographic examinations were performed, the finding of diverticulosis has been reported as 5.67 per cent in a series of 24,620 examinations[11] and as 8.5 per cent in a series of 47,000.[10] This correlates rather closely with the incidence of diverticula found at necropsy, reported by Rankin and Brown[11] as 5.2 per cent in 1925 cases.

It is difficult to ascertain the incidence of diverticulitis in relation to diverticulosis, but various writers report an incidence of 15 to 20 per cent, as determined by x-ray evidence and by necropsy.[10, 11]

It is rare to find diverticula or diverticulitis before the age of 35. After this age, the incidence of both increases steadily until at age 85, ⅔ of patients examined will be found to have diverticula.[14] The incidence of diverticulitis also rises steadily, and with advancing age, the incidence ascends sharply. Thus, in the sixth decade, approximately ⅕ of all patients with diverticulosis will be found to have radiographic evidence of diverticulitis, whereas in the ninth decade, this fraction increases to ⅛.[14]

Autopsy studies by Mailer[6] showed diverticula to be distributed throughout the colon in 30 per cent of the subjects with diverticulosis, and to be confined to the sigmoid in 40 per cent. However, clinical manifestations of the disease are almost always limited to the sigmoid. Colcock[2] reports diverticulitis to be confined to the sigmoid colon in 80 to 85 per cent of patients.

The findings that diverticulitis occurs more frequently with advancing age, and that diverticulitis is practically limited to the sigmoid, have led many writers to implicate mechanical factors as significant in the etiology of this disease. Some factors felt to be important are: narrow lumen, solid fecal stream, stasis, chronic constipation, increased intraluminal pressure in the sigmoid during defecation and loss of muscle tone.

SYMPTOMS AND DIAGNOSIS

The individual diverticulum is a small herniation of the mucosa through a defect in the muscular coat, often occurring along the border of one of the longitudinal muscle bands. A diverticulum of the colon is similar to other types of herniation in the gastrointestinal tract, in that, so long as there is free communication with the lumen of the colon, few symptoms are produced. However, if for any reason the neck of the diverticulum becomes occluded, an inflammatory reaction will occur, with resulting irritation and edema and further occlusion. The inflammatory process thus produced spreads by direct extension to involve the adjacent bowel wall, causing edema, induration, interference with normal peristalsis and irritability of the colon. By this process, the clinical symptoms of uncomplicated diverticulitis are produced.

Uncomplicated diverticulitis will usually respond to conservative medical treatment, consisting of a low residue diet, mineral oil or demulcent such as Metamucil, antispasmodics, sedation and antibiotics. In approximately 4 or 5 per cent of patients medical treatment fails.[3] In these cases the inflammatory process progresses and one of the surgical complications of diverticulitis ensues.

Progressive edema and induration may occlude the bowel lumen, resulting in partial or complete obstruction. The inflamed diverticulum may adhere to small bowel, bladder, or abdominal wall, and by the process of inflammation and necrosis, a fistula may result. A perforated diverticulum may be walled off and result in a localized abscess, or, less frequently, the walling off process may not be effective and a generalized peritonitis will result. Lastly, the inflammatory process within the diverticulum may result in gross hemorrhage. All of these complications will usually require surgical treatment.

Most patients with uncomplicated diverticulitis will give a history of recurrent episodes of cramping, left lower quadrant pain associated with constipation, gas and distention. At times there will be constipation, alternating with spells of diarrhea and the passage of mucous and pus, which is often associated with the relief of pain. In case of partial obstruction from edema and inflammation, some patients may notice a decreased caliber of the stool. With increasing inflammation, soreness and tenderness of the left lower abdomen are common. If an abscess develops from perforation of a diverticulum, a mass may be felt through the abdominal wall, or the rectum, or vagina. If the abscess encroaches upon the urinary bladder, frequency and dysuria will be noted. With the complication of sigmoidoenteric fistulas, diarrhea and frequent episodes of cramping, lower abdominal pain, along with soreness and tenderness, are common symptoms.

Bleeding, varying in amount from a slight streaking, or small clots, to gross hemorrhage, will occur in from 5 to 15 per cent of patients.[5] Rarely, the hemorrhage may be so severe as to necessitate emergency colectomy.

Patients in whom a sigmoidovesicle fistula develops will report dysuria, urgency, frequency, passage of gas and/or feces through the urethra. This complication occurs approximately five times more frequently in men than in women.[7] Apparently, the uterus acts as a barrier between the sigmoid and bladder, helping to prevent this complication in the woman.

Rarely, a diverticulum may perforate into the free peritoneal cavity, resulting in generalized peritonitis and its symptoms of sudden severe abdominal pain, along with direct and rebound tenderness and rigidity of the abdominal wall.

The diagnosis of diverticulitis by history and physical examination alone is not reliable. Roentgenologic examination in the form of a barium enema is the most valuable diagnostic aid. With this procedure, diverticula are seen as rounded, knoblike projections from the lumen of the colon and are most frequently noted in the descending and sigmoid colon. Roentgenologic signs of diverticulitis consist of spasm, edema, deformity and hypermotility. In cases where severe inflammation is present, spasm may be so marked as to cause a false filling defect and may completely occlude the lumen. A true filling defect may be produced by thickening of the bowel wall from edema or extrinsic pressure from a localized abscess and inflammatory reaction. A combination of these filling defects is commonly seen in most cases of diverticulitis and often leads to difficulty in differentiation of diverticulitis from carcinoma. Schatzki[12] has reported important points in helping differentiate these lesions. Spasm of the adjacent colon in diverticulitis is common, whereas with carcinoma the adjacent colon is usually normal. The mucosal pattern in the involved segment of diverticulitis is normal,

whereas in carcinoma the mucosal pattern is often destroyed. In diverticulitis, a long segment of bowel is usually involved, whereas carcinoma usually affects a short segment. The edge of the defect in diverticulitis is often cone shaped; in carcinoma, the edge is usually shelflike. The above differential points in some cases are atypical, so that differentiation in all cases is not possible. Schatzki[12] concludes that "accurate diagnosis is easy in most, difficult in some and impossible in a few."

Smith[13] reports proctologic examination to be of little value in making the diagnosis of diverticulitis. Its chief value is in helping to rule out the presence of carcinoma.

TREATMENT

The treatment of the complication of diverticulitis may be carried out in one, two or three stages, as the situation demands, and as sound surgical principles dictate. A general outline of these stages follows.

Stage 1. Proximal colostomy using right half of transverse colon. It is important to avoid placing the colostomy in the splenic flexure, descending colon, or sigmoid, since to do so would seriously interfere with later resection of the diseased area. Additional procedures, such as drainage of an abscess, or an area of a perforation, may be employed at this stage.

Stage 2. Resection of the diseased area of colon with end-to-end anastomosis. The segment of colon removed should be generous, in order to remove all gross disease, as well as to obtain healthy bowel for the anastomosis, and to prevent recurrent diverticulitis. At times, it will be necessary to include a portion of the descending colon, in which case, the splenic flexure must be mobilized in order to perform the anastomosis without tension. Some element of residual inflammation and edema will often be present and in many cases, diverticula will be present in or near the proximal line of resection. Thus, there is always the possibility that an inflamed diverticulum may be incorporated in the anastomosis, with resultant danger of leakage. For this reason, it is safer to have a proximal colostomy present during the healing phase.

Stage 3. Closure of colostomy. A barium enema should be performed 3 weeks following resection. If there is no extravasation of barium and no stricture formation, the colostomy may be closed safely. The colostomy should seldom, if ever, be closed without previously resecting the diseased area of colon; since to do so will in most cases result in a recurrence of complications.

There are seven indications for surgical treatment. These are perforation, with abscess formation; perforation, with generalized peritonitis; presence of a fistula; obstruction; hemorrhage; recurrent severe diverticulitis; and inability to differentiate diverticulitis from carcinoma.

The most common complication of diverticulitis is rupture of a diverticulum, with formation of a peridiverticular abscess (fig. 1). If the abscess is small when the patient is first seen, medical treatment may be tried, and is often successful. If the abscess subsides under medical treatment, the patient may later be treated surgically, the indication being severe diverticulitis. Patients receiving medical treatment must be followed closely, and unless the abscess subsides promptly, surgical treatment should be instituted without delay.

Initial surgical treatment consists of the performance of a diverting colostomy, utilizing the right half of the transverse colon. Most patients thus treated will respond by regression of the abscess, and subsidence of the inflammatory process. In the occasional case in which the

FIG. 1. Large peridiverticular abscess displacing sigmoid.

FIG. 2. Rupture of diverticulum with generalized peritonitis.

abscess does not subside, additional complications frequently arise. The abscess may rupture into small bowel, bladder, or rarely, through the abdominal wall. It may also enlarge, and by extrinsic pressure, cause small or large bowel obstruction. For these reasons, an abscess which fails to respond to medical treatment and proximal diverting colostomy should be drained, preferably using the extraperitoneal route, if possible. This procedure will frequently result in a sigmoidocutaneous fistula. However, with the presence of a proximal diverting colostomy, the fistula does not represent a serious problem and will often close spontaneously.

Both of these surgical maneuvers, that is, the performance of the colostomy, and the drainage procedure, are carried out as expeditiously as possible and with minimal trauma and manipulation. These patients are usually quite ill and withstand prolonged or extensive surgery poorly. The vast majority of patients thus treated will improve dramatically, with a rapid lysis of fever, return of appetite and increased sense of well-being. By these simple surgical procedures, infection is controlled and the stage is set for future definitive surgery.

The second complication of diverticulitis requiring surgical treatment is rupture of a diverticulum with generalized peritonitis (fig. 2). In approximately ¼ of these cases, premonitory symptoms will be absent[14] and, hence, the diagnosis of ruptured appendix, or ruptured duodenal ulcer, is likely to be made. Either of these erroneous diagnoses is likely to result in an incision through which exposure of the sigmoid may be difficult. If a right lower quadrant incision has been made, it can usually be extended toward the midline, and drainage of the sigmoid area accomplished. A second incision should then be made in the right upper quadrant for the performance of a diverting colostomy. In case an upper abdominal incision has been made, it can be used for the colostomy, providing that the transverse colon is pulled well to the left so that the right half of the colon is used. The degree of inflammation present makes it extremely unwise to attempt suture closure of the perforation or exteriorization of the sigmoid. In any event, the proper treatment of generalized peritonitis from a ruptured diverticulum consists of drainage of the area of perforation and performance of a right transverse colostomy. The principle of allowing the patient to recover and preparing for future definitive surgery is again utilized.

The third complication requiring surgical treatment is the presence of a fistula, either internal or external (figs. 3 to 5). In these cases, surgical treatment may be carried out in one of

FIG. 3. Coloenteric fistula with tract well outlined.

FIG. 4. Cystogram of patient with sigmoidovesicle fistual. Note roughening on posterior wall of bladder.

FIG. 5. Colocutaneous fistula

two ways. If there is considerable inflammation present, as evidenced by fever, tenderness, soreness, elevated sedimentation rate and leukocytosis, initial treatment should consist of right transverse colostomy, following which, the inflammatory process surrounding the fistula will usually subside, and occasionally, the fistula will close spontaneously. On the other hand, if there is minimal inflammation about the fistula, colostomy as the first stage of treatment may be omitted and, after proper preoperative preparation, resection of the sigmoid with closure of the portion of bladder or bowel involved may be carried out as the initial treatment. With this procedure, it is wise to perform a concomitant proximal colostomy.

Obstruction is the fourth complication requiring surgical treatment (fig. 6). Here, there is no alternative to a proximal colostomy, which again should be placed in the right half of the transverse colon. If there is a concomitant abscess, it may be partially responsible for the obstruction; if present, and after proximal colostomy the abscess does not subside promptly, it should be drained, as discussed above.

The fifth complication requiring surgical treatment is hemorrhage; being reported in from 5 to 15 per cent of patients.[5] Rarely, the hemorrhage may be massive and necessitate an emergency resection of the involved area. Usually, however, the bleeding is mild, and is noted as small clots or slight streaking. Other sources of bleeding, such as hemorrhoids, polyps, benign tumors, ulcerative colitis and carcinoma, may be responsible. Digital rectal examination, along with sigmoidoscopic examination and barium enema, will help make the correct diagnosis.

FIG. 6. Intestinal obstruction from diverticulitis.

FIG. 7. Barium enema illustrating the common problem of attempting to exclude the presence of carcinoma in a patient with diverticulitis.

With a history of rectal bleeding, the problem of differentiating diverticulitis from carcinoma becomes especially pertinent. Here, we find the sixth complication (or indication) requiring surgical treatment—that is, inability to differentiate diverticulitis from carcinoma (fig. 7). The problem arises because both carcinoma and diverticulitis occur frequently in the older age group and both have a predilection for the descending colon and sigmoid. Both produce similar signs and symptoms. It is extremely important to realize that persistent rectal bleeding is much more likely to be from carcinoma than from diverticulitis. Therefore, one should be very diligent in ruling out the presence of a carcinoma before attributing rectal bleeding to diverticulitis. If within reach of the sigmoidoscope, one may be able to visualize the lesion and obtain a biopsy. In most cases, however, the differentiation will depend upon roentgenologic findings, as seen by barium enema examination. After exhausting every effort, there will still be a number of patients in whom this differentiation cannot be made with assurance.[12] In these cases, the lesion must be considered as carcinoma, until proved otherwise.

Initial treatment in this situation should consist of a right transverse colostomy. A second barium enema performed some 2 or 3 weeks later may show changes from the initial examination which will help the radiologist make a more definite diagnosis. If carcinoma still cannot be definitely excluded, wide resection of the sigmoid and its mesentery should be carried out as soon as the inflammatory process has begun to subside. This will usually be from 2 to 3 weeks. It is well to realize that, even at laparotomy with the lesion fully exposed, it is often not possible to differentiate diverticulitis and carcinoma.[9] Therefore, a wide resection should be done and the removed specimen should be examined immediately by the pathologist. An end-to-end anastomosis is performed, with the colostomy being left open, to protect the anastomosis.

The seventh complication requiring surgical treatment is severe, recurrent diverticulitis. The current concept regarding treatment of this complication has been well covered by many excellent papers in the recent literature. Suffice it to say here, that severe, recurrent diverticulitis, occurring in a patient under 50 years of age, who has been on a good medical regimen, will often progress and result in the occurrence of one or more of the previously discussed complications. The occurrence of dysuria in a patient of any age is foreboding, since it often precedes the formation of a sigmoidovesicle fistula. The occurrence in the older patient of any of these additional complications, namely, perforation, abscess formation, obstruction, formation of fistula, or hemorrhage, represents a serious threat. It is now felt that patients under the age of 50 who develop severe diverticulitis, and patients of any age who develop dysuria, or who are disabled by frequent severe attacks of diverticulitis, should be treated intensively on a medical regimen until the acute inflammation subsides. Then, with proper preoperative preparation, resection of the sigmoid, with end-to-end anastomosis, can usually be accomplished as a one-stage procedure. Occasionally the anastomosis may prove to be difficult or insecure because of residual inflammation or edema, in which case, a proximal colostomy should be added.

After the initial treatment of the complications described above, the patient is treated with proper antibiotics, high protein diet, vitamins, and if necessary, iron.

Two factors will influence the length of the interval necessary between the first stage of treatment and resection of the diseased area:

(1) the amount of inflammation present and the speed with which it subsides; and (2) the degree of certainty with which carcinoma can be ruled out. If carcinoma can be definitely excluded, it is best to wait from 2 to 4 months. On the other hand, if carcinoma cannot be excluded, resection must proceed as soon as the inflammatory process has partially subsided. This is usually in from 2 to 4 weeks. If the inflammatory process subsides slowly, it suggests the presence of carcinoma.

The colostomy can usually be closed within 2 to 3 weeks from the time of resection and anastomosis. Before closure of the colostomy, a barium enema should be performed in order to ascertain the presence of a well healed anastomosis with an adequate lumen.

SUMMARY

The surgical treatment of the complications of diverticulitis has been presented in detail. Some of the ideas may seem "old-fashioned" or overly conservative. It is true that recent advances in the field of antibiotics and chemotherapeutics may shorten the intervals between stages; however, the sound and proved principles of colon surgery cannot be ignored. It is felt that proper staging of procedures will result in a decreasing incidence of morbidity and mortality.

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BLOOD BROTHERS—THE LERICHE SYNDROME AND CORONARY ARTERY DISEASE*

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Since Leriche in 1923, and subsequently,[3, 9] first called attention to the syndrome of localized obliterative disease of the terminal aorta which now bears his name, great strides have been made in the treatment of this severely incapacitating disease. Lumbar sympathectomy was first advocated for this condition and, even as late as 1955, Grimson and colleagues[5] advised a modified sympathectomy to include the lower thoracic ganglia for this condition, although these authors conclude that the relief of symptoms of claudication is better with grafting procedures.

Next came the advocation of thromboendarterectomy.[13] Although the results of this procedure were better than those obtained with sympathectomy, it still left much to be desired, particularly when dealing with the iliac and femoral arteries where the rate of reocclusion and thrombosis became higher the more distal the vessel operated upon.

Lastly, the use of arterial homografts and synthetic materials has come to the fore and the literature is legend with articles on this subject.[3, 6, 11] It is our feeling that, in general, better results are obtained with grafting procedures in dealing with the lower abdominal aorta and its major branches and that sympathectomy is probably the operation of choice in cases of arteriosclerosis obliterans where the involvement is mainly of the terminal arteries within the foot.

ETIOLOGY

The cause of atheromatous degeneration within blood vessels has been the subject of much clinical and laboratory investigation. Armstrong and associates[1] made a study of life insurance statistics and concluded that there is a definite relationship between obesity and arteriosclerosis. Pomeranze and associates[12] have shown that there is a relationship between a high blood cholesterol level and arteriosclerosis.

Having had a moderate amount of experience in treating occlusive disease of the terminal aorta and its branches, we were struck by the fact that a high incidence (about 50 per cent) of manifest coronary artery disease exists in these patients. Just what the incidence of subclinical coronary disease is, will have to be determined in the future. Curiously enough, the literature contains very few reports of this relationship. Julian and associates[7] and Massarelli and Estes[10] refer to degenerative diseases of the aorta as being a part of a generalized disease. DeBakey and associates[2] make an indirect reference to this relationship in a table giving the cause of 10 deaths following aortic resection—4 due to coronary occlusion and 3 from cardiac failure. In 1954 Faivre and associates[4] described "coronary trouble" in connection with "arteritis of the extremities."

One of us (T. W. I.) states that 50 per cent of the Leriche syndrome patients who came to the Cardiology Clinic have manifest heart disease. This close association of coronary disease and aortic occlusive disease is borne out by the following case reports.

CASE REPORTS

Case 1. A 49-year-old white man was admitted to the hospital complaining of claudication in the lower extremities upon exercise. One year previously he had had a left lumbar sympathectomy with good, but only temporary, results. Following evaluation, a freeze-dried homograft was inserted into the distal aorta and common iliac arteries with excellent immediate results as far as leg symptoms were concerned. The patient returned to the hospital, however, 14 months after surgery with rather extreme shortness of breath and retrosternal pressure and pain along the left arm upon exercise. These symptoms were relieved by nitroglycerin. Electrocardiograms were abnormal but revealed no definite pattern of infarction.

Case 2. This patient, a 49-year-old white man,

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complained of cramps in the legs and thighs and bilateral hip pain, on exercise, of 6 years' duration. One year after onset of symptoms the patient had a bilateral lumbar sympathectomy with good results for about a year. At that time his symptoms returned and were much more severe. Following evaluation, the terminal aorta and both common iliac arteries were replaced with a freeze-dried homograft with excellent results and complete relief of claudication. After 10 months the patient was readmitted to the hospital in rather severe congestive heart failure. An electrocardiogram at this time revealed an acute anterior myocardial infarction. Two months later, after a slow but good recovery, the patient had a second myocardial infarction from which he succumbed. Of particular interest is that at the postmortem examination this patient had a 1-cm. atheromatous plaque in the homograft.

Case 3. A 39-year-old white man, was admitted who, at the age of 30, noted onset of exercise pain in the calves of both legs which gradually progressed upward to involve the thighs. He also complained of progressive impotence. Past history revealed that his father died at 37 from coronary occlusion and that his mother was suffering from hypertension. No arterial pulsations were palpable below the umbilicus and aortograms revealed no filling of the aorta below the renal arteries (fig. 1). He was scheduled for surgery but 2 days before the scheduled operation, he had a severe coronary occlusion from which he almost succumbed. Recovery was slow but eventual. The occurrence of a severe coronary infarction in the face of a completely blocked aorta was considered an absolute contraindication to surgical correction of the Leriche syndrome and the patient was so informed. At the present time, 2 years after the infarction, the patient is alive and on active military duty and states that his claudication symptoms have improved. This is probably a result of self-limited activity since his infarction.

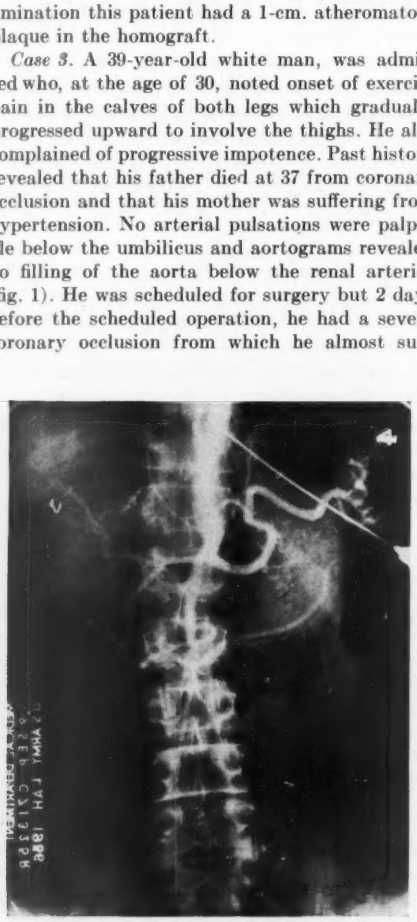

FIG. 1. Aortogram demonstrating complete occlusion of the aorta below the renal arteries.

Comment. One of the above patients developed angina pectoris following aortic grafting. Another patient had two episodes of myocardial coronary infarction following aortic grafting, the second of which was fatal. In the 3rd patient, operation was felt to be contraindicated following the occurrence of a severe coronary occlusion.

It is postulated that the cause of coronary symptoms following aortic grafting is a diminished coronary blood flow with some degree of myocardial ischemia following release of the aortic obstruction. The lower extremities, formerly deprived of an adequate blood supply after surgery, are now "stealing" a portion of their blood supply from the myocardium. Another factor which may cause symptoms of myocardial disease is the increased activity of the patient following relief of symptoms of claudication in his legs after surgery. These two factors, *i.e.*, decreased coronary blood flow and increased metabolic demands on the myocardium, are thought to be the prime factors in causing cardiac symptoms following successful correction of aortic occlusive disease. These factors may also operate to cause cerebral symptoms as illustrated by the following case.

Case 4. A 63-year-old white man entered the hospital with complete occlusion of the terminal aorta and no pulsations in either lower extremity. He had experienced severe symptoms of claudication in the legs, thighs and hips for 8 years as well as complete impotence. At operation, endarterectomy of the distal aorta and each common iliac artery was performed. The day following surgery, no pulses were obtainable in either extremity and it was felt that the patient had developed a clot in the terminal aorta which had extended down into the common iliac vessels. He was advised that another operation was necessary but refused permission. Twelve days later, still with no pulsations in either lower extremity, the patient was ambulated. Following this, he stated that his legs felt "like bags of water—dead" and requested another operation. At reoperation 2 days later

(14 days after the endarterectomy), a clot was found which extended from about 1 inch above the aortic bifurcation down to the profunda femoris branch of both common femoral arteries. A long homograft was sutured in place; from above the former arteriotomy site in the terminal aorta down to the common femoral artery in both thighs just proximal to the profunda branch. Following this the patient made a good recovery with bounding anterior and posterior tibial pulsation bilaterally. On the 5th postoperative day the patient was allowed to sit up and he complained of severe faintness and dizziness. Although these symptoms persisted for several weeks, they gradually subsided as his brain became slowly accustomed to its decreased blood supply.

CONCLUSIONS

Certain deductions may be made from the case histories given above:

1. Patients with so-called Leriche syndrome have generalized arterial atheromatous degeneration.

2. Relief of abdominal aortic obstruction results in a decrease in coronary and cerebral blood flow.

3. Relief of symptoms of a severe Leriche syndrome usually results in increased activity of the patient and therefore increased metabolic demands on the myocardium.

4. Claudication symptoms of patients with aortic obstruction may well act as the safety valve or the "governor on the carburetor" and in this way protect the myocardium.

5. Usually, patients with symptoms of angina pectoris, or those with a history of coronary infarction or cardiac failure, are not suitable candidates for aortic occlusive surgery. Indeed, operation is contraindicated in most cases of this nature.

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ESOPHAGEAL HIATUS HERNIA COMPLICATED BY ESOPHAGITIS*

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The treatment of esophageal hiatus hernia and at times the associated esophagitis offers problems not always easy of solution. The following case reports dealing with this condition may prove of interest to those concerned with these problems.

CASE REPORTS

Case 1. A Caucasian male, aged 61, had been known from history and x-ray examination (fig. 1) to have a sliding esophageal hiatus hernia for approximately 8 years. Symptoms had not been of sufficient severity to justify surgical intervention. However, in August 1957, he stated that he was having considerable substernal discomfort with indigestion and heart burn and that at times the passage of food seemed to meet an obstruction, resulting in vomiting. This was noted to occur particularly when swallowing meat. X-ray study at this time (fig. 2) showed the hernia to have increased somewhat in size, and to retain barium. There was no evidence of associated ulceration.

On September 6, 1957, a transthoracic repair of the hernia was done using the technique described by Allison,[1] and the patient made a good recovery. One week following the hiatal hernia repair the patient left the hospital but 2 weeks later he began to complain again of substernal discomfort and trouble in swallowing. X-ray examination at this time suggested the possibility of a recurrence of the hernia. There was an area of narrowing of the terminal esophagus suggesting edema but no evidence of ulceration. Esophagoscopy showed redness and edema of the mucosa of the distal esophagus without ulcer formation. Two months following the transthoracic repair of the hernia, because the patient's symptoms had increased in severity, he was explored transabdominally. The stomach was found to be well below the diaphragm without any evidence of recurrence of the hernia. A bilateral vagotomy and pyloroplasty was done. Following this procedure, the stomach failed to empty. Bile was regurgitated into the stomach but this organ seemed to be devoid of peristaltic activity (fig. 3). Nasogastric suction, intravenous fluids, blood and various smooth muscle stimulants were used without avail. The patient was kept in physiologic balance, as far as we could determine, by frequent blood serum investigations. On November 23, 1957, 18 days following pyloroplasty and vagotomy, a posterior gastroenterostomy was done. Following this, nasogastric suction and intravenous injections had to be continued intermittently for approximately 1 month before the stomach emptied adequately. The patient made a good recovery and has been relieved of his previous symptoms.

Case 2. A Caucasian male, aged 58, was first seen on May 21, 1958. He complained of epigastric distress, burning, and belching with occasional vomiting. These symptoms had been present for the past year with increasing severity. His symptoms were so distressing that he was incapacitated for work. X-ray examination of the upper gastrointestinal tract revealed a shortened esophagus with approximately 8 cm. of the stomach held fixed within the thorax with one small area suggestive of an ulcer crater (fig. 4); this was probably an acquired short esophagus and diaphragmatic hernia from repeated episodes of ulceration and fibrosis. The study otherwise was negative.

On June 6, 1958, a repair of the esophageal hiatus hernia was done. Adequate freeing of the terminal esophagus and reduction of the hernia by the abdominal approach was made difficult because of the patient's barrel-like physique; therefore, a transthoracic intercostal incision was added to the approach through the abdomen and a satisfactory repair was obtained in the usual manner. Bilateral vagotomy to reduce gastric acidity and a posterior gastroenterostomy for drainage of the stomach was then done. A double lumen tube was passed through the abdominal wall to the left of the upper part of the left rectus incision and into the stomach, the distal end being passed into the efferent limb of the gastroenterostomy, the proximal end being left in the stomach (fig. 5). Subsequently, suction was applied to the proximal tube and fluids were administered through the distal tube. On the 9th postoperative day, the double lumen tube was removed after x-ray study showed barium to be leaving the stomach without delay and the patient was eating a normal diet without discomfort (fig. 6). The patient went home on the 11th postoperative day, relieved of his previous symptoms.

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DISCUSSION

Esophagitis is the most common disease of the esophagus and may be due to a variety of causes. It is perhaps more frequently associated with gastric and especially duodenal ulcers than has been suspected. Hiatus hernia is the most frequent condition giving rise to esophagitis, and it is thought that this results from interference with effective closure of the esophagogastric juncture,

FIG. 1. Case 1. Hiatus hernia August, 1950

FIG. 3. Case 1. Failure of the stomach to empty and absence of peristalsis 2 weeks following bilateral vagotomy and pyloroplasty.

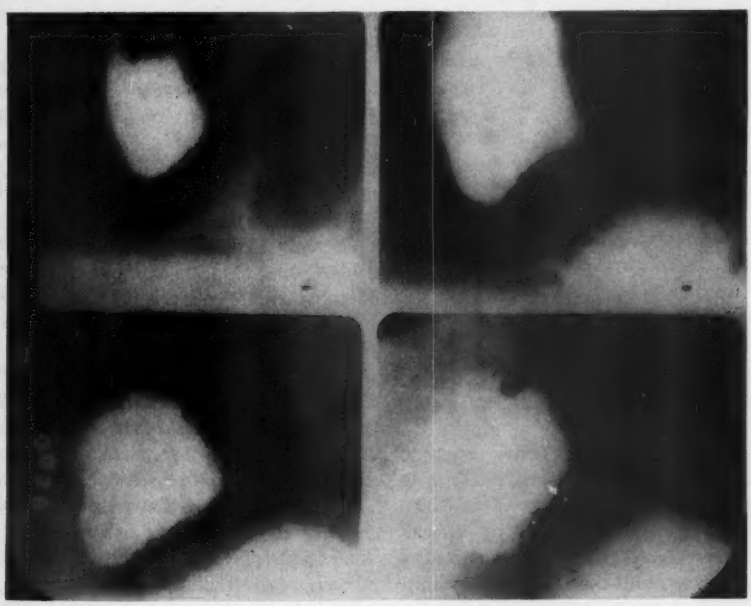

FIG. 2. Case 1. Increase in size of the hiatus hernia August, 1957

FIG. 4. Case 2. Hiatus hernia with probable ulcer crater.

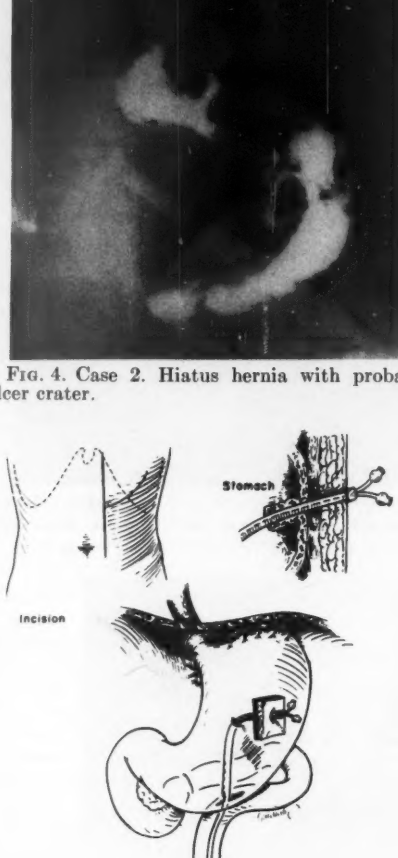

FIG. 5. Case 2. Incision, method of repair of the hiatus hernia, and the method of using the double lumen tube inserted through the abdominal wall.

allowing regurgitation of acid gastric contents into the esophagus. Any condition giving rise to regurgitation or vomiting is prone to produce esophagitis. It has been estimated by Vinson[7] that 10 per cent of benign esophageal strictures occurring in women result from nausea and vomiting of pregnancy. It is a well known fact that the

FIG. 6. Case 2. Barium leaving the stomach without delay on the 9th postoperative day.

squamous epithelium of the esophagus is very susceptible to the eroding effects of gastric juice, and Wangensteen and Leven[8] have stated that if all the gastric juice left the stomach *via* the esophagus, esophagitis and ulcer formation would in all probability be the most frequent of all clinical types of ulcer.

The treatment of esophagitis has occupied the interest of many observers because its symptoms and resulting complications have been difficult to combat in many cases. Conservative measures without correcting the inciting cause have in most cases been unsatisfactory. Therapy used in treating duodenal ulcer medically has in general been found useful in treating esophagitis; namely, a bland diet, antacids and anticholinergic drugs. If the inciting cause is an esophageal hiatus hernia, reduction of intraabdominal pressure by losing weight is sometimes found to be of value as well as having the head of the patient's bed elevated so that the hernia tends to reduce itself during sleep. Strictures of the esophagus resulting from ulceration with resulting scar tissue formation may be relieved by esophageal dilation. It is generally believed that adequate repair of an esophageal hiatus hernia will relieve the symptoms and prevent progression to ulceration, fibrosis and stricture, if this has not already occurred.

In 1949 Wangensteen and Leven[5] published their observations in 6 cases in which they had done a high gastric resection with ensuing favorable results even though ulceration, fibrosis and stricture of the esophagus had occurred. In 3 patients, it had been associated with duodenal ulcer. One patient had a gastric ulcer, another had a congenitally short esophagus and the third had a stricture of the lower esophagus of unknown origin. Further use of this method by Wangensteen and Leven apparently has been satisfactory. They attribute the favorable results of high gastric resection for esophagitis to the reduction in gastric acidity and the quickening of gastric emptying time.

Merendino and Skinner[6] have approached the problem by interposing a jejunal segment following gastroesophageal resection. This apparently has worked satisfactorily in the experimental animal and in a relatively small number of human beings.

Before the American Surgical Association in April 1956 Burford and Lischer[2] reported on the treatment of the short esophagus complicated by diaphragmatic hernia and esophagitis by Finney pyloroplasty. Improvement had occurred in 15 of the 16 cases so treated and in 10 patients virtually complete relief had been obtained. It was the consensus of the discussers that in all probability most of these cases were not congenital, but resulted from contracture of the esophagus from inflammatory reaction. In discussing this paper, Clagett said that the results at the Mayo Clinic in treating this condition had generally been unsatisfactory. He commented further that their results had been encouraging in about 10 cases in which they had done a transthoracic resection of the esophageal stricture, uniting the cardia to the esophagus, followed by resection of the antrum of the stomach with gastroduodenostomy. However, Clagett thought that simple pyloroplasty as described by Burford and Lischer would be far preferable, due to its simplicity, if it survives the test of application. In April 1958 Ellis, Andersen and Clagett, in an unpublished paper before the American Surgical Association, stated that they had now operated upon 25 cases of short esophagus with stricture in which esophagogastrectomy, antral excision and bilateral vagotomy had been employed with excellent results in the majority.

Merendino and associates[5] presented a paper before the April 1957 meeting of the American Thoracic Association, "An Experimental Appraisal of the Finney Pyloroplasty in the Prevention of Esophagitis Following the Heller Myotomy." They made the following observasions: "It has been observed, both clinically and experimentally, that operative procedures which compromise the sphincter action of the esophagogastric juncture are attended by a significant incidence of postoperative esophagitis. Dogs subjected to Heller myotomy developed esophagitis in 70 per cent of cases, but when this was combined with Finney pyloroplasty, the incidence dropped to 9 per cent. Under these same circumstances, the addition of vagotomy to a pyloroplasty decreased the incidence of esophagitis but vagotomy alone increased the severity of the esophagitis."

The combining of vagotomy with pyloroplasty, or gastroenterostomy, has not been tried often enough and the results have not been observed sufficiently to draw conclusions as to this method of preventing or treating esophagitis. From the very limited experience recorded here, this method seems to have merit and is deserving of further trial. Theoretically at least, it would seem desirable to add vagotomy to some type of drainage operation in the treatment of esophagitis because of its well demonstrated value in decreasing gastric acidity.

Temporary gastrostomy, as advocated by Farris and Smith,[3] Gilchrist[4] and others is undoubtedly of great value in the postoperative care of such patients. While I have seen no permanent harm resulting from transnasal gastric suction, it would seem most undesirable to further irritate an inflamed esophagus when gastric decompression is indicated and can be accomplished by another method that has been found to be safe, efficient and certainly far more comfortable for the patient.

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IDIOPATHIC CHYLOTHORAX: REPORT OF A CASE AND REVIEW*

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The occurrence of milky-white, nonpurulent pleural effusion was described by Bartolet[1] over three centuries ago. The collection of chylous fluid in the pleural space has been known as an infrequent complication of trauma since 1663.[14] Nontraumatic chylothorax is caused most commonly by malignant neoplasms. Tuberculosis involving the pleura or the mediastinal lymph nodes, thrombotic occlusion of the left subclavian vein, filariasis and benign neoplasms[16, 27] are other causes. Chylothorax, due to unrecognized operative injury of the thoracic duct, has been noted much more frequently with the increase in the number and extent of intrathoracic surgical procedures.[15] It seems less well known that chylothorax may occur rarely without apparent causative trauma and in the absence of related neoplastic or inflammatory disease. Despite its rarity the possibility should be noted in order to help dispel the specter of obscure neoplastic disease after adequate search has revealed no apparent cause.

It seems improbable that rupture of the thoracic duct occurs spontaneously in the absence of some type of stress. Meade[18] has postulated a prior, localized fixation of the thoracic duct by neoplastic or inflammatory disease as a necessary prerequisite of spontaneous chylothorax. Hyperextension of the spine may then rupture the duct. Even such common stress as coughing may be followed by chylothorax. A so-called latent period sometimes exists between rupture of the thoracic duct and the subsequent escape of the chylous fluid from the mediastinum into the pleural space. The difficulty of relating some prior, mild or unremembered trauma to the subsequent appearance of chylothorax after a considerable latent period is readily apparent. Fears that the unexplained appearance of chylothorax in a young patient might be caused by occult neoplastic or chronic inflammatory disease led to this review of the subject.

* Read at the meeting of The Southern Thoracic Surgical Association, Miami Beach, Florida, November 28-30, 1958.

CASE REPORT

An 18-year-old white schoolgirl was admitted to Tallahassee Memorial Hospital on December 11, 1957. Gradual onset of discomfort in the right shoulder, slight nonproductive cough and progressive dyspnea and weakness had been noted 1 month before. Her physician† found physical and roentgenographic evidence of a large pleural effusion on November 27 and removed 2 L. of chylous fluid from the right pleural space. An equal amount of fluid was removed on December 2 after which her chest roentgenogram was normal. Her shoulder pain persisted, dyspnea gradually returned and fluoroscopic examination on December 10 showed recurrence of the effusion. Repeated questioning revealed only that she had fallen while rollerskating 1 year before. She suffered no injury, continued to skate and noted no ill effects. She could remember no other falls, injuries or unusual physical activity. The past history and family history were unremarkable.

Physical examination revealed an apprehensive, dyspneic girl of normal development. Temperature was normal. Blood pressure was 110/70 mm. Hg. Pulse rate was 100 per minute. There was physical evidence of massive pleural effusion on the right with displacement of the heart and mediastinum to the left. The other physical findings were within normal limits. Blood examination disclosed 4,350,000 red cells per cc., 7700 white cells per cc. with 79 per cent segmented neutrophils, 1 per cent stabs, 17 per cent lymphocytes, 2 per cent monocytes and 1 per cent eosinophils. Hemoglobin was 87.5 gm. per cent. The urinalysis was unremarkable.

Closed intercostal catheter drainage rapidly emptied the right pleural space of 4 L. of typical chylous fluid and her dyspnea was relieved. By the following morning an additional 500 cc. of fluid had collected and drainage then ceased. A chest film on December 13 indicated complete reexpansion of the right lung with no pleural fluid. Thereafter intermittent drainage occurred at intervals of up to 36 hours. The fluid varied in degree of milkiness from time to time; later it became somewhat thicker and formed a fibrin clot on cooling. During the periods of no drainage the catheter was kept patent and fluoroscopy indi-

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cated no collection of pleural fluid until December 23 when a fluid collection was incompletely evacuated despite manipulation of the catheter. She was discharged to spend Christmas at home and returned on December 26. A chest film showed further increase in the pleural fluid which could not be evacuated through the catheter. On December 27 the right pleural space was opened through the 7th intercostal space and 1200 cc. of chylous fluid and fibrinous clot were removed. This made a total of more than 14 L. of chylous fluid removed during her treatment. The lung was partially encased in fibrinous clot of variable thickness. It was adherent to the diaphragm from which it was freed by finger dissection. The diaphragm appeared normal except for the fibrinous coating. The lung was easily decorticated. The parietal and mediastinal pleurae appeared thickened, edematous and hyperemic. Before operation 2 ounces of olive oil had been injected through a tube passed into the stomach. No leakage of chyle was seen along the mediastinum or elsewhere despite a careful and prolonged search. The thoracic duct was found between the azygos vein and aorta at the diaphragm without difficulty. It was tied twice with cotton. No accessory duct was found. The splanchnic nerve was identified. The wound was closed with one catheter draining the pleural space. Minimal serosanguineous drainage ceased within 12 hours but the catheter was not removed until the 2nd day. Her subsequent recovery was uneventful and she was discharged from the hospital on January 2, 1958. Her weight at this time was 110 pounds, representing a loss of about 15 pounds since the onset of the illness. She has remained well and a chest film made in October 1958 was normal.

DISCUSSION

Judging from published accounts, chylothorax resulting from any cause is uncommon and cases of undetermined etiology are rare. A complete review of the literature was not possible but only 3 instances of unexplained chylothorax in adults were found.[5, 9, 18] Reviews by Brescia[3] in 1941 and Stirlacci[23] in 1955 noted 8 and 5 cases, respectively, which were spontaneous in infants from 4 days to 13 months in age. In 3 instances[7, 13] the only known trauma was coughing and in 2 other patients chylothorax followed the exertion of diving and acrobatics,[18] respectively. In a single instance chylothorax was associated with a spontaneous pneumothorax.[4] Excellent reviews of the subject of chylothorax, both traumatic and nontraumatic, are available in publications of the past 20 years.[7, 13, 22, 27] The remainder of the discussion is confined to some of the pertinent points of diagnosis and treatment.

Diagnosis

The clinical findings of the patient with chylothorax are due either to the pressure of the intrapleural fluid collection or to the nutritional results of the loss of quantities of fluid, fat and protein and are therefore nonspecific.

Laboratory findings. The milky-white character of the chylous fluid obtained by thoracentesis should immediately suggest the diagnosis. Chylous fluid is quite variable in character depending upon its fat content and admixture with blood and serum, however, and one is cautioned about confusion with chronic empyema and other chronic pleural effusions which may contain cholesterol.[12] It seems unlikely that anyone would mistake chylothorax for a chronic empyema when copious milky fluid is aspirated from the pleural space repeatedly and frequently. Chyle in the newborn is clear fluid until the infant ingests food and fat, so that recognition of the true nature of the effusion may be delayed.[23] Similarly, it is known that the patient who is maintained on parenteral feeding without fat has only limpid fluid in his thoracic duct.[18]

Microscopic examination of chylous fluid reveals the fat globules which are stained by lipophilic dyes such as Sudan III. The milky character of chylous fluid may be cleared by shaking it with ether which dissolves the emulsified fat. The cells found in chyle are almost entirely lymphocytes, and examination of the patient's blood may reveal a paucity of lymphocytes when the loss of chyle is excessive. The fluid is bacteriostatic[13] and cultures of it are sterile. Despite contrary statements the fluid may coagulate on cooling as was observed in the case above.

Persisting confusion concerning the nature or origin of supposed chylous fluid may be settled by feeding the patient fat and a lipophilic dye and later recovering the dye-stained fluid from the chest. The green dyes (D & C no. 6) are recommended as more readily recognized in fluid which is mixed with blood.[12]

Treatment

The objectives of the treatment of chylothorax are the same whatever its etiology; namely, (1) cessation of the loss of chyle; (2) restoration of pulmonary function; and (3) protection of the

patient's nutritional status during treatment. Simple thoracentesis with complete evacuation of the fluid, repeated at intervals as necessary, is often effective. It is postulated that the production of pleural symphysis seals the area of leakage. If thoracentesis is ineffective or is required too frequently, closed intercostal catheter drainage of the pleural space is indicated. There is disagreement as to how long such conservative measures should be used before resorting to direct attack on the duct.[15]

When treatment becomes prolonged the nutritional support of the patient assumes increasing importance. The merit of reducing the fat content of the diet is controversial. Drinker[6] found that fat ingestion altered the fat content of chyle but did not change its volume of flow. A similar observation was made by Wessel[26] in an infant with chylothorax. Peters[19] stated more recently that following a meal containing fat the flow of chyle, which is highly lactescent, increased rapidly. Klepser and Berry[12] have demonstrated the engorgement of the thoracic duct which follows a fatty meal. Only a portion of the fat absorbed from ingested food is found in the chyle of the thoracic duct,[2] and it has been suggested that the amount of fat in the diet of the patient with chylothorax should be increased to compensate for that lost in the effusion.[15] Since the protein content of chyle may be almost as great as that of plasma, loss of protein may be of more nutritional importance than the loss of fat or fluid. In most instances the patient's diet probably plays no great role in the control of his chylothorax provided that he receives adequate protein, carbohydrate and fluid and provided that ineffective conservative measures are not unduly prolonged.

When repeated thoracenteses or adequate catheter drainage of the pleural space fail to stop the leakage of chyle or when complete evacuation of the pleural space is not attained, transpleural ligation of the thoracic duct is indicated. This procedure had been suggested and even attempted previously but was first successfully done by Lampson[13] in 1946. Its effectiveness and safety were shown by several subsequent reports.[7, 9, 12, 17] This procedure may be very simple yet because of the extremely variable anatomy of the thoracic duct and the difficulties of its identification, surgical control may be difficult or impossible at times. Probably only one relationship of the duct is constant in its course from the cisterna chyli to the left subclavian vein; its location on the anterior surface of the vertebrae between the azygos vein and the aorta at the diaphragm.[25] Even here the duct may be double despite numerous contrary observations.[10, 24] The splanchnic nerve, located in the same area, has been mistaken for the duct by competent surgeons. Such anatomic vagaries as are found in the adult are readily understood in the light of the embryologic development of the duct, which initially forms as paired ducts from the cisterna chyli passing cephalad along the aorta with numerous intercommunications. The right duct crosses to the left, dorsal to the aorta, at the 4th thoracic vertebra and finally empties into the left subclavian vein.[8] The embryonic left duct usually becomes obliterated.

Various measures have been employed to accentuate the duct before its surgical exposure and to demonstrate accessory ducts and branches during dissection. Preoperative administration of a fatty meal, with or without lipophilic dyes, may cause distention and allow easier recognition of the duct.[12] The subcutaneous injection of Evans blue dye into the thigh has been advocated for rapid delineation of the duct at operation.[11] At times none of these measures is helpful[9] perhaps owing to administration of the fat-dye meal too late in the preoperative period or perhaps owing to alteration of normal physiologic processes by anesthesia. Stranahan and coworkers[24] have described operative thoracic ductography in which the duct is cannulated and injected with radiopaque material, roentgenograms of the area are made, and then lipophilic dye is injected to allow visual identification of any accessory branches of the duct.

Although the thoracic duct may be demonstrated more easily when approached through the right pleural space it may also be controlled from the left (as it was in the first two successful cases).[13, 17] When the chylothorax is unilateral approach should be from the affected side in order that any needed decortication may be done. Various plastic procedures advocated for control of leakage from the duct are unnecessarily complex in execution[21] and accomplish no more than simple ligation.

The results of the treatment of chylothorax are dependent upon the etiology. The mortality of traumatic chylothorax has dropped from 45

to 10 per cent in 10 years[7] and the associated injuries contribute to this rate. Although little permanent benefit may be expected in the patient with chylothorax secondary to malignant neoplasm, considerable palliation may be afforded at times by ligation of the duct.[12, 17] The treatment of nontraumatic chylothorax due to benign pathology should give excellent results. All 3 adults noted above with unexplained chylothorax recovered following ligation of the duct. Of the 13 cases noted in infants 9 recovered following repeated thoracenteses. In 3 of the 4 infants who died, the thoracic duct could not be found at autopsy[23] perhaps due to congenital hypoplasia or absence although this was not stated.

SUMMARY

Chylothorax occasionally occurs in the absence of neoplastic or inflammatory disease and without known trauma. Such a case of idiopathic chylothorax is reported. The minimal degree of trauma necessary to cause chylothorax and the latent period sometimes existing between the rupture of the thoracic duct and the manifestation of the effusion may explain such idiopathic cases. Prompt evacuation of the pleural fluid by needle or catheter often allows healing of the thoracic duct. When this fails the duct should be exposed and ligated in the chest at the diaphragm. The morbidity and mortality of such treatment is now minimal.

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MALDESCENT OF THE TESTIS

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INTRODUCTION

In spite of all that has been written on the subject of maldescent of the testis there is no uniform opinion as to either the best method or the best time for treatment. Much conflicting advice is given. Many parents of boys with this condition seek several opinions and have their confidence destroyed by the different opinions presented to them. With this in view it was felt that a review of the subject, with an attempt to evaluate the significance of the work on the damage seen in the testis which is not normally descended, would be of value. In order to fairly evaluate the best methods of treatment it is necessary to review the whole subject since there is much confusion between facts and fancies. It will become apparent that in many respects the extent of ignorance is far greater than that of knowledge. It is because of this that opinions vary so much. But there are known facts and these should be kept clearly in mind in formulating opinions. Maldescent is not a single clinical entity but includes five different conditions. Unless these are kept in mind discussion becomes meaningless.

NORMAL DESCENT

Many of the present difficulties arise from the mystery which surrounds the normal descent of the testis into the scrotum. The testis extends from the diaphragm to the site of the future abdominal ring at 8 weeks of fetal development. Subsequently, due to differential growth rate and its remaining attached with its lower pole "tethered" at the abdominal ring, it comes to lie in the iliac fossa at or near the internal ring by the 7th month. It has not migrated but the other structures have moved relative to it.[51, 52]

From this intraabdominal position the testis descends to the scrotum during the next 2 months so that by birth its normal position is regarded as being in the scrotum. It is this stage of the descent that is not understood. At one time it was concluded that the gubernaculum pulled the testis down but this view then became unpopular. However, there is reason to think that it may play an important part. The gubernaculum is a multitailed bundle of fibromuscular tissue with much of its "fibrous" tissue gelatinous, primitive, nucleated tissue. The various "tails" described run to the dartos of the scrotum, medially to the pubis and fascia over the rectus muscle, laterally into the area between Scarpa's fascia and the external oblique, and inferiorly to the perineum and the fascia by the saphenous opening. It is along the lines of these "tails" that the testis descends, either normally into the scrotum, or abnormally in ectopia to one of the other sites. Ectopic testes are never found in areas to which "tails" of the gubernaculum have not been described. If the testis is withdrawn from the scrotum in an infant during a hernia operation its accurate replacement may be difficult if it is pushed down from above. However, if traction is exerted on the bottom of the scrotum it quickly slips back to its normal position. This indicates that there is a structure present adequate to pull the testis down.

It has been said that the testis is squeezed out of the abdomen by the internal oblique muscle. Though it cannot be said that this plays no part, it can at best only push the testis to the external ring.

The cremaster muscle is also said to be responsible and is associated with gubernaculum both by Hunter[27] and by Curling.[10] Lewis[30] attempted to show the influence of the cremaster by cutting the genitofemoral nerve (its nerve supply) in rats. When done under 8 days the testis on that side did not descend but when done after descent had occurred the testis remained either intraabdominal or scrotal, whereas the opposite one was migratory. How far this evidence can be applied to man remains to be decided.

At the time of normal descent the anterior abdominal wall is closing and the intestines are migrating back into the abdominal cavity with a probable increase in intraabdominal tension.

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Provided there were a hernial sac (the processus vaginalis) present, this pressure would tend to force the testis, lying at the internal ring, toward the bottom or scrotal end of this process. That this can happen is seen in female infants where the ovary is not infrequently found in an inguinal hernia at operation.

This raises the question whether the processus vaginalis is first formed as a sac into the scrotum, into which the testis normally descends, or whether the descending testis draws the processus down with it. Arey[1] states that at the beginning of the 3rd month while the testes are still fairly high in the abdomen, saclike pockets appear which become the processus vaginalis. At operation the processus is sometimes found to extend well beyond the testis and so it seems fair to conclude that the processus is formed first.

Finally it must be mentioned that at the time of normal descent the maternal circulation and the fetal circulation contain a high concentration of chorionic gonadotropins. There is no proof that these exert any influence on the descent but there are some suggestive facts. In Macacus monkeys, whose testes normally descend at puberty, these hormones given before puberty bring about descent.[16] There are many reports in the literature of the efficacy of treating boys with undescended testes with chorionic gonadotropins, particularly when there is hypogonadism.[12] When there is no hypogonadism some cases still descend at least coincidentally with hormone therapy.[13, 46, 47]

It is thus possible in summary to postulate that in the normal male fetus between the 7th and 9th month the gubernaculum shortens and the testis descends to the bottom of the processus vaginalis by its traction under either nervous or hormonal stimulation. At the same time the testis is pushed by the increased abdominal pressure, squeezed by the internal oblique and milked by the cremaster. It is impossible as yet to prove which of these factors is important or indeed whether they play any part at all.

ETIOLOGY OF MALDESCENT

Because of the ignorance of the mechanisms of normal descent, the etiology of maldescent cannot be fully known. However, it is possible to list certain factors which are known to play a part and to discuss others that may be influential.

Hypogonadism is often mentioned as a cause but what is meant is hypopituitary secretion, either generalized or of gonadotropins. Heller and Nelson[24] have pointed out that it is not possible to diagnose hypogonadism until puberty and difficult even then. If chorionic gonadotropins, transferred from the maternal circulation, were responsible for normal descent in late fetal life, it does not seem reasonable to blame lack of function of the child's pituitary for something it is not responsible for. Yet it has been suggested that the cases of maldescent which respond best to exogenous chorionic gonadotropins are those in which there is inadequate endocrine secretion from the pituitary. There is a type of pituitary dwarfism seen clinically which has abdominal retention of the testes as one symptom. Clinicians recognize that the "fat boy" type with quite marked and often localized pelvic obesity may have bilateral undescended testes. The proof of pituitary endocrine dysfunction in these cases is difficult because the normal excretion of gonadotropins before puberty is so low. In these cases a therapeutic trial of chorionic gonadotropins may be the only way of establishing a diagnosis. It is difficult to conceive how hormonal factors can be responsible for unilateral maldescent. If there is satisfactory hormonal stimulation to bring one down, why not both? If "hormones" are to be implicated the receptor organ must be to blame in these cases.

The testis itself can be shown to be abnormal in some cases. This abnormality may be gross such as abnormal separation from the epididymis,[31] or abnormal length of the mesorchium. It may also be histologically abnormal with complete disorganization of the normal tubular structure. Such a picture is quite different from the lack of tubular maturation and the peritubular fibrosis seen with the "normal" testis which is undescended after puberty.[8, 17, 44] It is distinguished as primary testicular dysplasia. It is assumed to be a congenital maldevelopment unassociated with the position of the testis. Although it may well be found more commonly in testes which have not descended, there is no proof that this histologic primary dysplasia is of itself an etiologic factor, since it may also be seen in descended cases.

Ectopia could have two causes. It might be because the gubernaculum is abnormal with overdevelopment of one of the accessory tails instead of the scrotal one; or the way into the scrotum could be anatomically blocked causing the testis to follow one of the other tails.

Certainly anatomic blocks could cause failure of descent. Abnormal development of the inguinal canal has been blamed, and failure of the "third inguinal ring" of McGregor[34] at the entrance to the scrotum has been implicated. The testis may also be held up by adhesions. I have operated on one case in which the inguinal canal was normal with a hernial sac extending to the top of the scrotum. Preoperatively the testis was not palpable but on exploration it was found at the internal ring. While attempting to mobilize the testis a firm fibrous cord was found attached to the upper pole. Further dissection revealed this to be the appendix. There had been no history of appendicitis or any symptoms suggestive thereof since birth.

It must be admitted that the foregoing discussion is begging the question. Other than the fact that on rare occasions an anatomic obstruction to normal descent can be shown, there is no completely proved etiology known.

Because of this lack of knowledge of the normal mechanisms and their aberrations it is difficult to devise an incontrovertible plan for treatment. There are four general modalities used: watchful waiting, endocrine therapy, orchidopexy and orchiectomy. Each has its own enthusiasts but if certain other facts are taken into consideration it is possible to show that all have their place and that they should be used having careful regard to the type of case and the desired objective.

Three general points must be considered before discussing a rational plan of therapy: (1) the classification of the type of maldescent; (2) the effects of maldescent on the individual; (3) the reason for treating.

CLASSIFICATION

1. Secondary cryptorchidism. This is used to describe patients where the cryptorchidism is a feature of a more generalized disease. It is typified by pituitary dwarfism. Its recognition is important since the patient, not the cryptorchidism, requires treatment primarily.

2. Primary testicular dysplasia. As already mentioned, this term is used to describe cases where the histologic structure of the testis itself is disordered. The tubules are difficult to identify, as such, and there is little or no similarity with normal testicular structure. The fault must lie in the failure of normal differentiation and development at a very early stage in fetal life. Unfortunately, although this condition may be suspected because of the abnormal size and feel of the gonad, it can only be proved by biopsy. Therefore, this diagnosis cannot be made clinically. It does influence the results of therapy because such a gonad cannot achieve normal function no matter where it is.

3. Incomplete descent. This term is used to describe the cases in which one or both testes have not descended far enough, though they remain in the correct line of descent. They must be distinguished by careful examination from the ectopic and the retractile testes. A fact that is not emphasized enough is that a testis which lies in the inguinal canal is not palpable. In the young child with generous suprapubic fat it is impossible to differentiate a testis in the canal from an intra-abdominal testis. As the child gets older a vague sense of a mass may be detected and pressure on this area may give typical testicular pain but it cannot be clearly outlined. If the testis is "emergent" it can be coaxed out of the canal by medial "milking" when the patient is sufficiently comfortable and relaxed.[4] It then becomes palpable. It is now important to note whether the testis as it is milked out of the canal moves downward and medially toward the scrotum, or upward and laterally away from the scrotum (figs. 1 and 2). In the latter case it is one of incompletely descended ectopia. If the testis is without the inguinal canal and situated suprapubically it

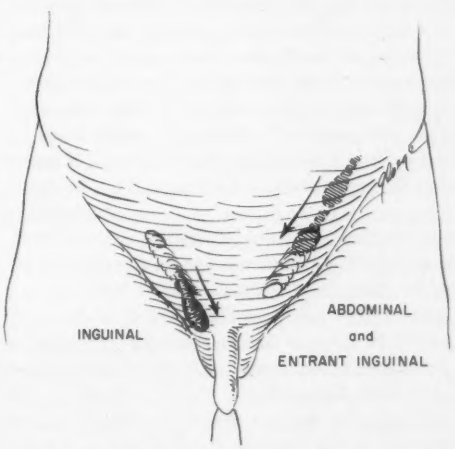

FIG. 1. Incomplete descent. On the left are shown the positions which are never palpable. On the right is shown the emergent inguinal position after the testis has been "milked" out of the inguinal canal.

FIG. 2. Inguinal ectopia. On the right is shown the emergent inguinal ectopic testis which is not palpable until it is "milked" out of the inguinal canal when it is noted to turn laterally. On the left is the inguinal ectopic which is easily palpable because it lies superficial to the external oblique fascia.

must be decided whether it is incompletely descended or retracted. In the former case the scrotal sac will be inadequately formed. Observation of the scrotal development is a most important distinguishing feature in these cases. Scorer[40] points out that the diagnosis is easier in the newborn because of the lack of fat.

4. Ectopia. The ectopic testis is one which has descended but has descended to the wrong place. It is important to search carefully, not only over the inguinal canal where the ectopic testis is so commonly found, but at the base of the penis, over the saphenous opening and in the perineum. Also, as mentioned above, the testis must be observed as it is milked out of the canal if found therein. Superficial inguinal ectopia, *i.e.*, the testis lying on the outer surface of the external oblique over the internal inguinal ring (fig. 2) and emergent superficial inguinal ectopia account for almost all cases of ectopia. Louw[32] found 40 per cent of his cases were ectopic, and of these, 27 out of 29 were of the inguinal type. It must be pointed out that many authors consider ectopia very much less common. This is because they do not recognize the fact that the palpable testis at the site of the internal inguinal ring is ectopic. Unless it is superficial to the external oblique this structure effectively prevents accurate palpation. It is astonishing that this diagnostic error should be so prevalent when the truth is so easily demonstrated at operation.

The importance of the accurate diagnosis of ectopia, complete ectopia and incompletely descended ectopia, lies in the fact that the treatment is no longer controversial. There is no alternative but a plastic operation to redirect the line of descent.

5. Retractile. The retractile testis causes confusion because the extent of the scrotal sac[4, 5] and the power of the cremaster in infancy are both underestimated. In infants the testis can easily be manipulated up over the pubis into an inguinal pouch which seems an extension of the scrotal sac. If a large series of infants is observed and the cremasteric reflex illicited, it can be seen that in a number the testis is drawn completely up out of the scrotal sac so that it becomes difficult to palpate. This reflex is much more sensitive in some than in others so that in these highly sensitive infants it is rare to find the testis in the scrotum. Repeated examination is necessary, using adjuncts to produce relaxation, before the fact that the testis is merely retracted can be demonstrated. The best illustration of the importance of appreciating this fact may be seen in the series of Einhorn and Rowntree[13] who had to reject 50 per cent of cases referred to them as errors of diagnosis. Also, Hamilton and Hubert[21] diagnosed "Pseudocryptorchidism," *i.e.*, retraction only, in over 60 per cent of cases referred to them.

It is this confusion regarding the diagnosis that makes the interpretation of results so exceedingly difficult. This aspect will be alluded to again during the discussion of therapy. Before discussing therapy it is pertinent to discuss the reasons for any therapy at all.

EFFECTS OF MALDESCENT

It is dangerous to lump all cases of maldescent together in discussing the adverse effects on the individual but it is difficult to discuss each type separately because so little is known. No adequate classification is standardized. Furthermore, in cases of incomplete descent relatively few authors outline the degree of incompleteness. Some work has been done but insufficient for any final conclusions to be drawn.

1. Impairment of spermatogenesis. There is a great deal of evidence that spermatogenesis is adversely affected in a testis which is not in the scrotum. The animal experiments of Wangen-

steen[39] show that placing a normal testis in the abdominal cavity or in the abdominal wall causes degeneration. This change is reversible if it is not allowed to proceed too long.

Bilateral cryptorchidism is reputed to be associated with complete infertility but this is questioned by some. Uffreduzzi[49] claims that 10 per cent may have some spermatozoa in the semen at least for a few years after puberty.

Some work has been done studying the histology of "retained" testes in various age groups. While these studies are not all in agreement, most of the recent work starting from that of Southam and Cooper,[45] and of Cooper[9] in 1929 and including that of Engle,[17] Nelson,[37] Sohval[44] and Charny and his co-workers[7] allows the following conclusions to be drawn. Some testes show primary dysplasia from the beginning. Others and probably the majority show very little change from the normal for the first few years of life and are not markedly abnormal until the first signs of puberty. However, from 6 years on there are gradually increasing changes apparent which become marked between 9 and 11 years. These changes at first consist of little more than thickening of the membrana propria. Then there is a lack of cellular development of the tubules with peritubular fibrosis though Sertoli and Leydig cells remain normal. With sexual maturity the picture is one of considerable abnormality of the tubules, there being only Sertoli cells recognized. Again, the Leydig cells remain unaltered. Cooper suggests that these changes are less marked the further that the testis had descended. Charny does not confirm this but he does not say to which groups the testes that he has been biopsying belong.

In an effort to confirm the relative lack of damage before the age of 8 and 9 and its increase thereafter, I have been biopsying all testes at orchidopexy. Figures 3 to 9 are microphotographs of my last 7 cases.*

Figure 3, case 1, aged 5, shows left emergent inguinal testis with the processus vaginalis extending well into the scrotum. The vas was found to extend to the bottom of the processus and then up to the testis. The right testis was normally descended.

In case 2, aged 6, (fig. 4) the testis was found at the internal ring at operation. It could not be palpated preoperatively. The other testis was normally descended.

Figure 5, case 3, aged 8, shows the left emergent inguinal ectopic testis; right testis normally descended at birth. The patient had received treatment with chorionic gonadotropic hormone.

Figure 6, case 4, aged 10, shows the left inguinal ectopic testis; right testis normally descended.

Figure 7, case 5, aged 11, shows the right emergent inguinal ectopic testis; left testis normally descended.

Figure 8, case 6, aged 11, shows the right emergent inguinal ectopic testis; left testis normally descended.

In case 7, (fig. 9), the patient, aged 12½, had had an operation for hernia on the right side 1 year previously. It was not possible to discover whether the testis had been normally descended before this but the scrotum on that side was hypoplastic. The left testis was normally descended. The right testis was lying outside the external ring in an ectopic position but was bound there by scar tissue from the previous surgery.

The biopsy specimens from these cases have been reviewed by Mostofi.[36] He states that case 7 (fig. 9) shows, except for the thickening of the tunica, essentially normal development of the seminiferous tubules compatible with the age of 12. The other six biopsies show varying degrees of fibrosis of the interstitial tissue and of the tunica; most prominent in cases 3 and 4 (figs. 5 and 6), least in cases 5 and 6 (figs. 7 and 8). The seminiferous tubules are lined by Sertoli cells; germ cells being rare or absent. The interstitial cells consist of small spindle cells which show no evidence of activity. The changes in all but case 7 are interpreted as those of congenital gonadal dysgenesis and secondary fibrosis. Case 2 (fig. 4) is considered inadequate for satisfactory interpretation.

Of these 7 consecutive cases, 6 have testes which appear to be congenitally abnormal. If this were a true picture of the incidence of primary testicular dysplasia, treatment aimed at achieving subsequent spermatogenesis would be useless in the majority of cases. Since MacCollum[33] and Gross and Jewett[20] have achieved fertility in 75 to 80 per cent of patients after bilateral orchidopexy various possibilities must be considered. This may be an unusual and atypical series. The interpretation of these biopsies may be incorrect and their visible abnormality be the result of their

* Cases 1 to 6 were operated on in Children's Hospital, Washington, D. C. and the pathologic studies were made by Drs. Rice and Guin.

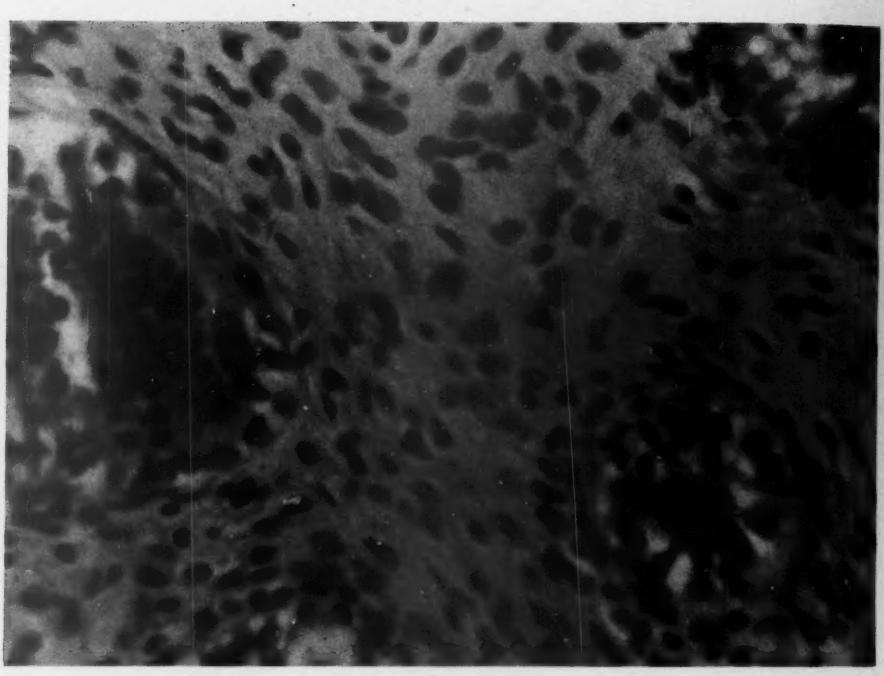

FIG. 3. Testis, aged 5; emergent inguinal, × 390

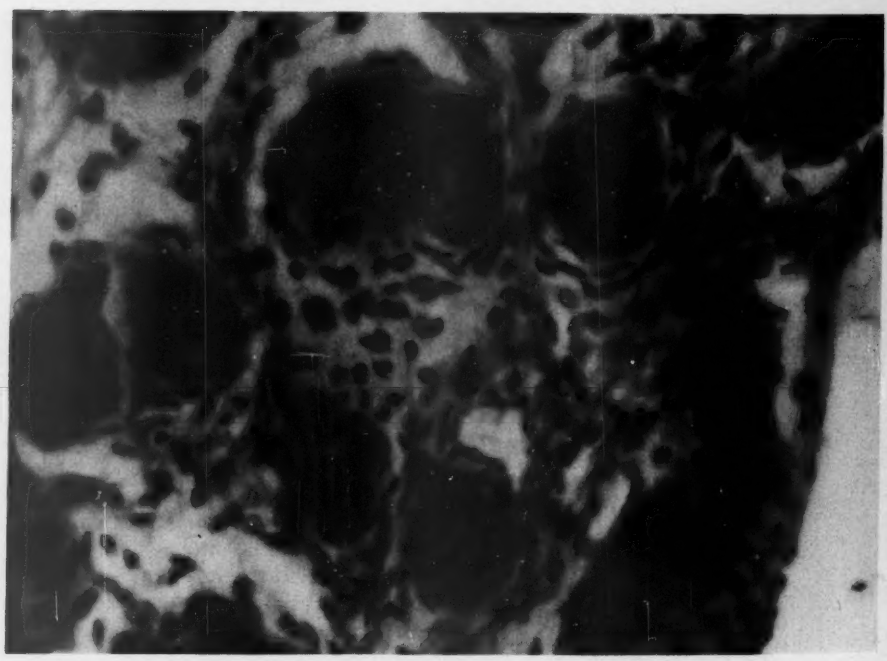

FIG. 4. Testis, aged 6; entrant inguinal, × 390

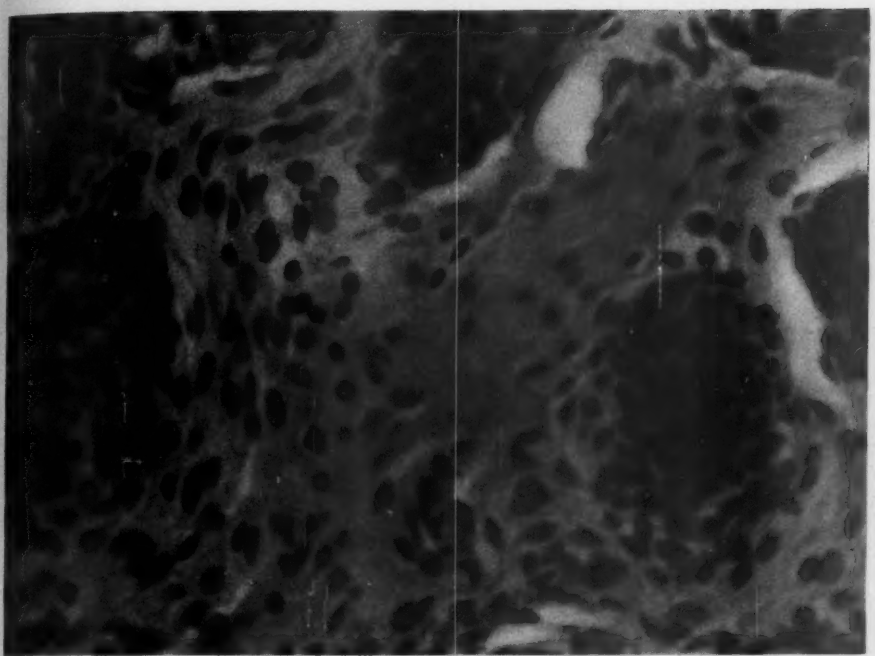

FIG. 5. Testis, aged 8; emergent inguinal ectopic, × 390

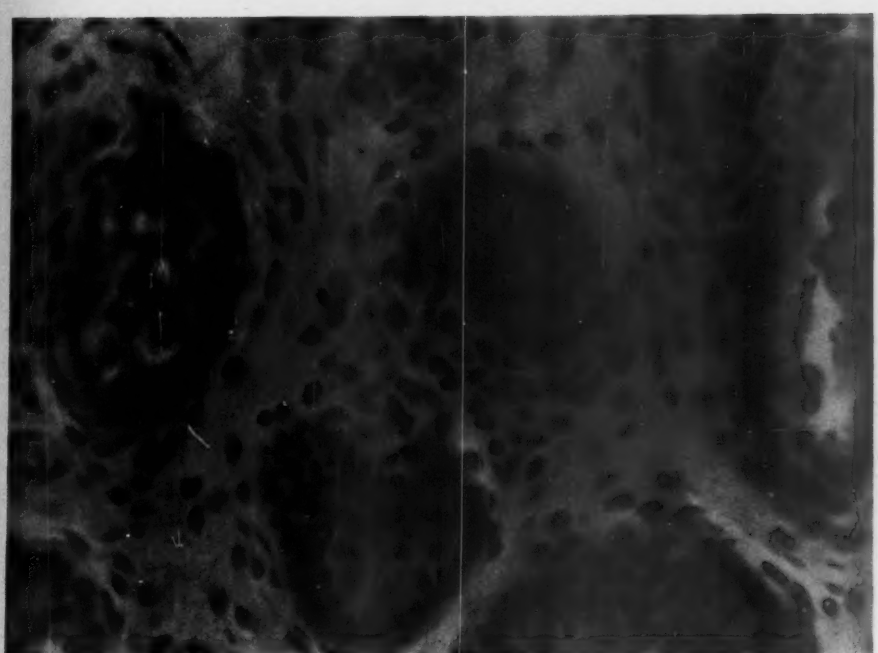

FIG 6. Testis, aged 10; inguinal ectopic, × 390

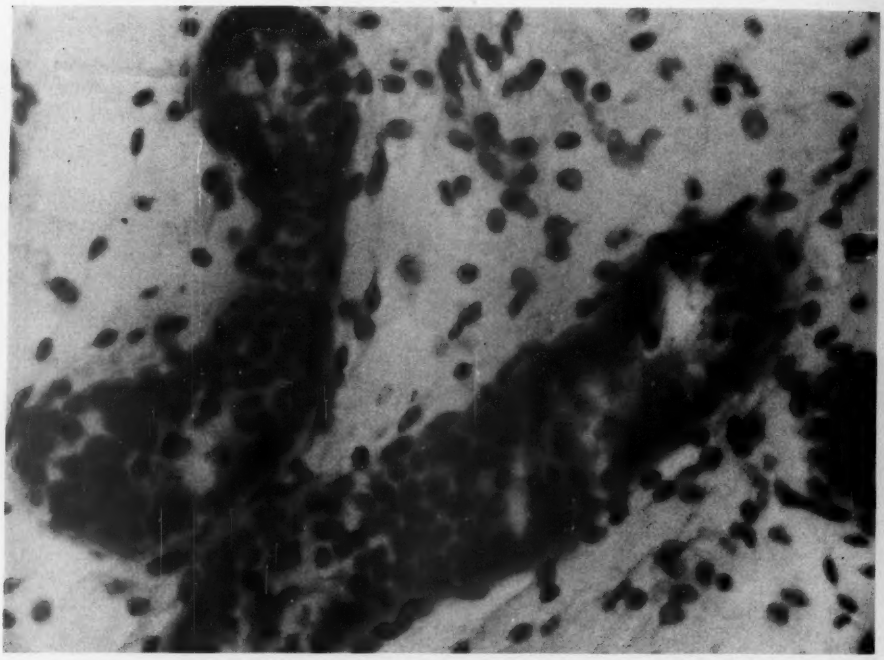

FIG. 7. Testis, aged 11; emergent inguinal ectopic, × 390

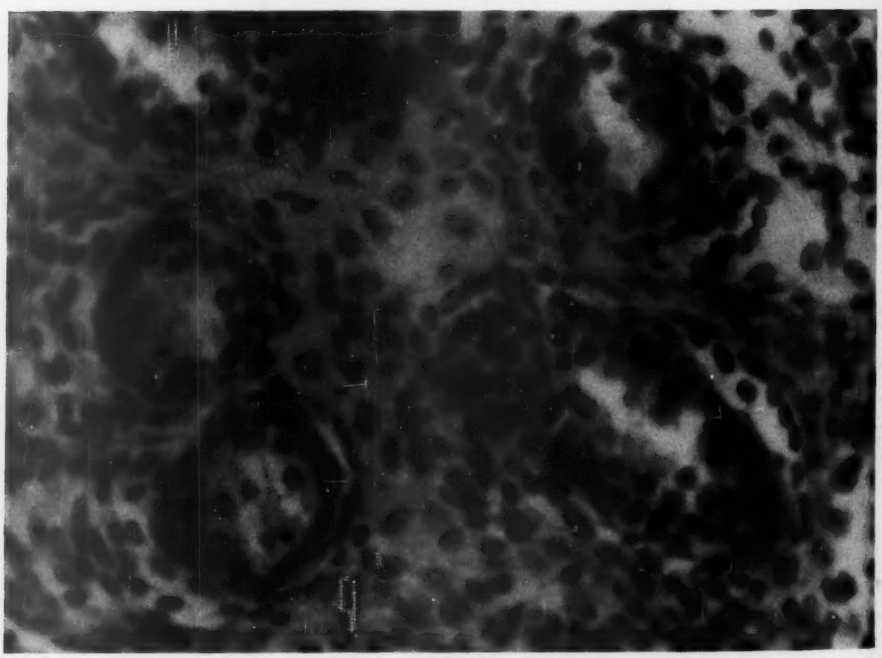

FIG. 8. Testis, aged 11; emergent inguinal ectopic, × 390

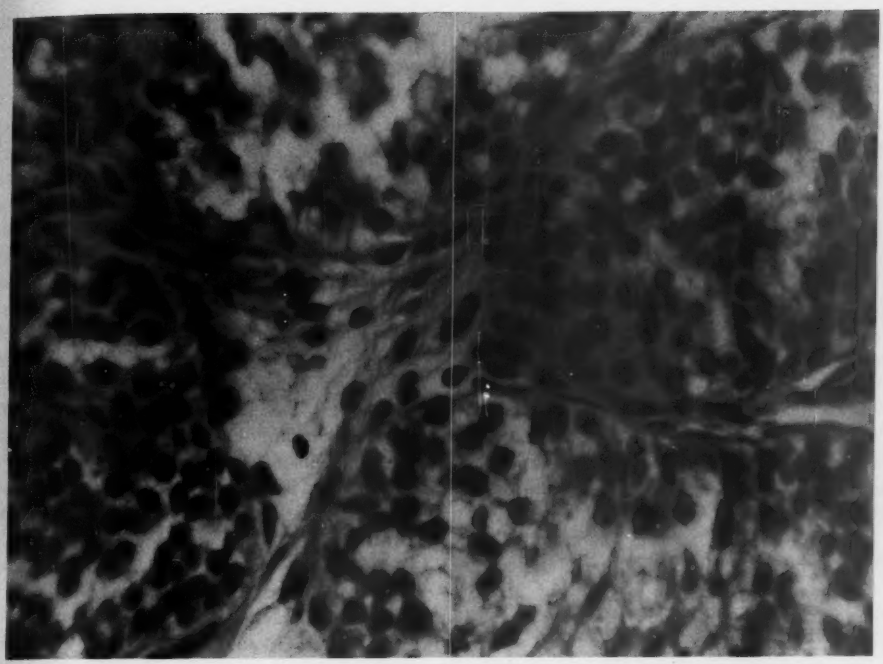

FIG. 9. Testis, aged 12½; inguinal ectopic after herniorrhaphy (see text), × 390

maldescent. If this is so, the damage is earlier than, more severe than and rather different from that usually reported. The changes seen may be reversible after orchidopexy. The extent of abnormality seen in these testes is best appreciated by comparing them with the normal testes at similar ages shown by Charny and Wolgin.[8]

These testes may have failed to descend because they were abnormal. No biopsy was taken from the normally descended testis in any case. It would be interesting to know whether the other testis was normal in structure as well as position. Hansen and Charny disagree about the effect of unilateral cryptorchidism. Hansen,[22, 23] studying sperm counts in men with unilateral cryptorchidism, some treated and some untreated, found that about 33 per cent had an inadequate count and these he considered infertile. This implies that the other normally descended testis in these 33 per cent was abnormal. In most of the remainder of his cases the sperm count was approximately half normal. Charny and Wolgin[8] say that the incidence of sterility among unilateral cryptorchids is the same as that for the normal male population.

2. Diminished endocrine function. The unilateral cryptorchid shows no clinical manifestation of diminished function and neither does the bilateral unless it is a case of secondary cryptorchidism or unless misguided treatment has caused testicular atrophy. However, Engberg[15] measured the endocrine output of these cases and found that it was definitely decreased even with unilateral cryptorchidism.

3. Liability to trauma. It is not tenable to suppose that intraabdominal retention increases this liability, but in cases where the testis lies just outside the external ring and over the pubic bone or in the perineum this is the real factor. The latter cases often complain of pain and this is sometimes the presenting symptom. The undescended testis is relatively fixed and cannot avoid trauma in the way that a scrotal one will.

4. Association with hernia. Between 75 and 90 per cent of cryptorchids have failure of obliteration of the processus vaginalis.[18, 35, 49] Not all of them have any clinical evidence of hernia in early life. Some authors maintain that this is no indication for operative treatment, while others disagree. It would be interesting to follow a series of

"late spontaneous descent" to see if there is an increased incidence of hernia above that of the rest of the population in later life.

5. Torsion of testis. Here it is important to distinguish between extravaginal torsion, which shortens the cord as it twists and gives the appearance of an undescended testis, and intravaginal torsion. The latter is probably more common because maldescent is associated with a higher incidence of gross deformity of the testis and epididymis. This deformity often takes the form of either a separation of the two organs or a long mesorchium. It does not seem frequent enough to be a justification for treatment.

6. Incidence of malignancy. Much has been written about this and the conclusion to be drawn from the evidence so far produced is that malignancy is about 50 times as common in undescended testes.[3, 50] The intraabdominal testis is about 4 times as likely to become malignant as one outside the inguinal ring. There is no evidence that orchidopexy alters this increased incidence of malignancy.[19, 30] The incidence, though greater than in normally descended testes, is still far less than the incidence of malignancy in the female breast. It would be more logical to do prophylactic mastectomies than orchiectomies.

7. Psychologic effect. The psychologic effect of testicular maldescent on an individual is very variable. There can be no doubt that it causes psychologic trauma but there is marked variation both in the amount of this trauma and in the response of the individual. It is not an aspect of the problem that should be lightly dismissed. Parental concern may make for a feeling of inadequancy in the individual or concentrate an unwarranted amount of attention on the genitalia. At school "friends" are unashamedly critical of any variation of the normal and implication of "something lacking" may lead either to withdrawal or compensatory aggression, and to excessive masturbation after puberty. There is no other defect which causes such a marked subconscious feeling of lack of normal virility. For psychologic reasons it would be desirable to treat in such a way that both testes are in the scrotum of all boys before school age. However, the psychologic aspects of treatment must also be borne in mind. Psychiatrists feel that any surgery before 5 or 6 is regarded by the child as further injury, not repair, and that operation on the genitalia is regarded as punishment. Thus psychic scars may be caused by treatment as well as by the defect.

REASON FOR TREATMENT

It is in the light of these effects of maldescent that the reasons for treating and the age at which therapy should be undertaken must be discussed.

In patients with secondary cryptorchidism the underlying condition must be treated when diagnosed and it is not intended to discuss this.

Since patients with primary testicular dysplasia can only be diagnosed by biopsy they cannot influence opinion. It should be remembered that they will influence results. If orchidopexies are performed without biopsy and then followed for subsequent spermatogenic function of the testis, any series containing a number of these cases must of necessity have poorer results.

Patients with pseudocryptorchidism need only correct diagnosis. The inaccuracy of diagnosis which is, unfortunately, quite common makes for much better results with either watchful waiting or hormone therapy than is justified.

In the remaining two groups, those with incomplete descent and those of ectopia, the primary consideration is usually directed toward preserving spermatogenesis. Since one normal testis is all that is required for normal fertility the object of improving the spermatogenic function in the maldescended testis in cases with a unilateral abnormality is not so important. Hansen[22] is the only author who reports subfertility in unilateral cryptorchids and the result of orchidopexy in his cases was so poor that he gives a very depressing outlook. However, if the normally descended testis becomes damaged by infection or trauma it is an advantage to have the other fully functional. Its spermatogenesis should be preserved if at all possible even if only to have it as a spare. This would indicate that treatment must be successful before the testis is irreparably damaged by its abnormal position. The increased incidence of malignancy is often the next consideration. Even if it is considered that the increased incidence to 50 times is exaggerated, it remains true that a neoplasm arising in a cryptorchid is symptomless until secondaries appear, when it is too late for any curative treatment to be undertaken. On the other hand, successful treatment resulting in scrotal testes does not prevent the subsequent development of neoplasia.[19] Neither the fear of torsion or trauma is considered of major im-

portance. The exception to this is the case with perineal ectopia. In this rare condition the presenting symptom, in my experience with these cases and in several reported cases, is pain on riding a bicycle. Parents of these patients are most insistent on early, in fact immediate, surgical treatment.

The most neglected aspect influencing treatment is the psychologic. Depending on the sociocultural environment of the patient this takes on different importance. In the American boy going to public school little attention will be attracted to the subject until exposure is inevitable in junior high school. In private school attention may be directed to the defect much earlier. However, it may be that the defect will be commented on at home as it was in one case of mine where the patient was tormented by a younger brother. In England where more boys go to boarding school at 7 or 8 it is highly desirable to have the defect rectified by this time.

TREATMENT

1. Watchful waiting. It is considered by many that there is a sufficiently high incidence of spontaneous descent after birth to merit a long period of observation. Statistically it seems conclusive that spontaneous descent does occur. Bishop[3] states that roughly 42 per cent of premature males have maldescent, 10 per cent of full term infants, 2 per cent at puberty and 0.2 per cent adult males; Hofstätter[25] gives similar figures. Although these figures are widely accepted, Scorer[40] examined 2700 consecutive males at birth and found 4 per cent of undescended testes in infants over 5½ lb. birth weight and 36 per cent when the birth weight was under this figure. He followed these cases and of the 37 full term cases found in the first 1000 deliveries only 6 remained undescended at the end of the first year of life. Of the 18 prematures only 1 remained undescended at the end of the first year. This gives a total of 0.7 per cent of cases of undescent at 1 year. This figure is comparable with the average percentage found in World War I recruits from Scotland and Austria and with figures from the American War Department and from Manchester quoted by Southam and Cooper.[45] On this basis Scorer claims that no testes descend spontaneously after 1 year of life. In contrast, Johnson[28] studied 544 cases and followed them from 7 to 17 years of age. Of these, 300 cases descended spontaneously and the peak incidence of descent was between the ages of 11 and 13. Surprisingly, this is the age at which hormone therapy is reported to achieve the best result. It is pertinent to consider two points in these statistics. (1) How many pseudocryptorchids are included? (2) Is the late descending testis normal? The first question is unanswerable; even the most careful observers make some errors in diagnosis. One case was diagnosed after being seen by a general practitioner and two other experienced surgeons. At operation when the patient was anesthetized both testes were noted to be reposing in the bottom of the scrotum.

Studies of the histologic damage occurring in maldescended testes show that there is steady damage occurring increasingly rapidly as the development of puberty progresses. Charny's biopsies offer proof that in some cases at least, there will be severe damage by the time the testis does descend.[8] Furthermore, a great deal of psychologic trauma may have been done to the patient by this time.

In the light of the histologic studies and because of the psychologic aspects, watchful waiting should be classified as obstinate inactivity since its only proved efficacy is in cases of pseudocryptorchidism when time corrects the diagnostic error.

2. Hormone therapy. Both chorionic gonadotropins and androgens have been advocated for use in treatment; however, androgens are certainly dangerous. They cause the changes of puberty to a marked extent and may even precipitate premature fusion of the epiphyses. They also cause suppression of tubular function in adults with a subsequent rebound phenomenon.[24, 42] What effect they have before puberty has not yet been fully proved. Chorionic gonadotropins are most commonly used. The experimental work on monkeys by Deming[11] and by Engle[16] showed that these hormones bring down the testis before puberty; however, the normal time of descent in these monkeys is at puberty. In that the administration of these hormones brings on the changes of puberty it is not surprising that they produce descent in these monkeys. It does not follow that they will have the same effect in the human whose testis should be descended at birth. Reports of the clinical efficacy of hormones vary. Thompson and Heckel[46, 47] report that 20 and 24 per cent of a large series of undescended testes came down

after hormone therapy and concluded that only those which would have descended at puberty without treatment came down. Einhorn and Rowntree,[13] treating 40 cases which had been carefully selected to exclude pseudocryptorchids and those with "migratory testis," reported 14 successes. Of the 26 unilateral cases, 6 descended; of the 14 bilateral cases, 6 descended on both sides and 2 on one side only. Ten of their successes were between the ages of 8 and 13. Since this is the age group in which Johnson reported approximately 30 per cent of his cases descended spontaneously, 30 per cent success rate with hormone therapy does not seem encouraging. Bishop,[3] who advocates chorionic gonadotropin therapy, states in the course of his extensive review that this hormone (1) enlarges the testis, (2) stimulates the connective tissue elements associated with the testis in its descent and therefore elongates the structures of the cord and (3) stimulates the secretion of androgen, thus causing the full development of the sexual apparatus and assisting the descent of the testis. There is little proof that any of these statements is true. As an example of the confusion that obfuscates the subject, some of the results he quotes should be noted. Out of 25 cases of "canalicular" testes, by which he means those which he claims to be able to palpate in the inguinal canal, 24 came down with treatment. He had no success with cases classified as "canalicular impeded." The clinical difference between what he calls "canalicular" and "canalicular impeded" seems rather nebulous and would be difficult to diagnose on examination. Of 46 cases with "extra canalicular" testes 31 came down, but he states that some of these may have been "retractile." He admits that it is difficult to distinguish between "impeded," "deviated," "delayed," and "retractile" cases.

Another thought is raised by the work of Charny and Wolgin[8] in which some biopsies of testes undescended after hormone therapy and some that descended after a course of treatment are shown. In these cases the degree of peritubular fibrosis and tubular immaturity and damage is rather startling. The pictures in these cases resemble the damage seen in much older boys, in late puberty, where the testis remains undescended. This is logical enough if it be accepted that chorionic gonadotropins stimulate the changes of puberty both in the patient generally and the testis specifically. Consequently, giving this hormone has the identical effect of waiting until puberty in the hope that spontaneous descent will occur. Most authors are agreed, that after puberty it is too late to obtain a good spermatogenic result. Is not the same then true after a course of chorionic gonadotropin?

However, there is another unknown to be considered. What degree of testicular damage is reversible? How far is the peritubular fibrosis seen in these cases a reversible change and how much does it prevent subsequent tubular maturation? Although the weight of opinion is that most of the changes are irreversible, there is one conflicting report by Rea[39] who performed a two-stage orchidopexy on a man aged 20. Biopsy at the first stage showed severe damage and no spermatogenesis. The second biopsy 6 months later at the second stage showed the tubular epithelium much improved in appearance. The patient was accidentally killed 22 months after this and was shown to have spermatozoa in the tubules. In animals Wangensteen[50] showed that placement of a normal testis in the abdominal cavity or wall prevented spermatogenesis and ultimately caused atrophy. However, if this was not allowed to proceed too long the change was reversible by returning the testis to the scrotum.

What then is the place for hormone therapy? It is used to replace watchful waiting by the impatient. It is used in cases with "hypogonadism" if this can be diagnosed. It is used before surgery just to see what will happen. It is used before surgery "to make the operation easier." Since in this case it increases the metabolic activity of the testis and therefore its sensitivity to lack of blood supply it is really contraindicated before the trauma of surgery. Except in cases of secondary cryptorchidism it should be regarded as a "sop to Cerberus" and probably a poisonous one.

3. Orchidopexy. A remarkable number of operations have been devised for placing the testes in the scrotum. It is not intended to describe them all in this discussion. The vital point is whether or not surgery is a satisfactory method of treatment and if so, when it should be done. First it may be said that it is the only method of treatment suitable in cases of ectopia. In cases of ectopia the surgery is relatively simple when the testis is fully descended to an ectopic position whether this be inguinal, penile, perineal, or femoral, because the cord structures have sufficient length. In the emergent inguinal ectopic cases the

surgery may be difficult because of the inadequate length of the blood vessels.

In cases of incomplete descent the merits of surgery must be weighed against watchful waiting, hormone therapy and even orchiectomy. The disadvantage of the first two methods has already been discussed. If by orchidopexy a normal size, normal consistency testis could be guaranteed in the scrotum after treatment it would be better than the other methods. However, this is not so. Though surgeons themselves reporting on their results are usually favorably impressed,[48] others such as Charny[8] and Hansen,[23] examining a number of postoperative cases from various sources, are most unimpressed. Charny[8] writes, "Surgical trauma has possibly been more deleterious than the inimical environment of the inguinal canal." Charny and Hansen are more concerned with spermatogenesis than cosmetics. Unfortunately few surgeons have followed their cases from that point of view. It is hard to do so because only about 20 per cent of the cases are bilateral. If a simple follow-up study is done to check fertility unilateral cases must be discarded since the other testis will provide sufficient spermatozoa for fertility. If unilateral cases are followed useful information depends upon subsequent biopsies. Ideally, there is need for a study where serial biopsies are performed both before and following treatment. Until such a series is available there is no firm ground for conclusions because, in contradistinction to the reports of Charny and Hansen, MacCollum,[33] Hotchkiss,[26] and Gross and Jewett[20] report approximately 80 per cent fertility in bilateral cases. A possible reason for the difference of findings and opinions lies in the actual technique of surgery.

There are two problems to be overcome; the first is to get the testis down far enough to lie in scrotum without tension on its blood supply; the second is to keep it there postoperatively. It is because of these that most of the techniques of orchidopexy have been described. Bevan[2] suggested cutting the spermatic vessels if they were too short and relying on the external spermatic arteries' anastomotic circulation to maintain the testis. These vessels are inadequate. In fact, the demands of the testis for its blood supply are exacting. In dogs, Smith[43] showed that spermatogenesis was impaired by 2 hours' clamping of the vessels and eliminated after 6 hours. After 10 hours' occlusion the testis atrophied into a fibrous mass. Keetley[29] and Torek[48] suggested that the testis could be fixed down by attaching it through the bottom of the scrotum to the thigh. Ombrédanne[38] attached it through to the contralateral side of the scrotum. Cabot[6] suggested traction and this has been applied in many ways with many different devices. However, the spermatic artery is a long vessel with most of its course from the aorta retroperitoneally.

It has been pointed out by numerous authors since Torek that extensive retroperitoneal freeing of the vessels will give extra length. Since the course of these vessels is from the midline laterally and then medially through the internal ring toward the scrotum, it seems logical that moving it medially in its course and allowing it to come through the posterior wall of the inguinal canal directly would help.[18, 30] By dividing the inferior epigastric vessels this can be done but the gain may be disappointing since moving the vessel medially removes part of the lateromedial bend but increases the anteroposterior curvature.

In these extensive dissections there is real danger of damaging the artery. In the dissection the "suspensory ligaments" of Browne[5] and Eisenstaedt[14] become very apparent. They are worth re-emphasis since their division gives more increase in length than any other single maneuver. They extend from the endopelvic fascia to the internal spermatic fascia and seem most marked on the lateral side of the spermatic artery internal to the internal inguinal ring. After opening the canal and the internal ring they may be seen by putting gentle medial traction on the testis and cord. Damage to the veins is seldom considered but rough handling may cause venous thrombosis with resulting impairment of blood supply and marked congestion of the testis. Traction on the testis at operation can be seen to cause the veins to go into spasms. Any form of anchoring the testis either by fixation to the thigh or by traction with tape or elastic bands is absolutely contraindicated if it puts any tension on the blood supply of the testis. To put tension on a pedicle contravenes the most elementary principle of plastic surgery. Either the cord and vessels must be so freed without damaging them or the vas that the testis will lie easily in the scrotum or it should not be put in the scrotum at all. It should be brought down as far as it will comfortably lie and be left there for a year. Then a second operation will usually succeed in bringing it the rest of the way.

The usual reason given in defending methods using fixation or traction is that these devices are used to prevent subsequent retraction of the testis upwards. If the testis lies easily in the scrotum there is little that can pull it up though it may be squeezed out of the scrotum by the action of the dartos. In this respect it must be noted that the scrotum on the maldescended side is hypoplastic. It is necessary as a first stage in the operation to form a sac into which the testis can be placed. If the testis, as it is placed into this sac at operation, is slipped under a bridge of fascia over the pubis at the neck of the scrotum,[5] this will effectively stop subsequent retraction. If this is not considered satisfactory it would be logical to put traction on the scrotum or sew the scrotum to the thigh but not the testis itself. It is probably the fact that the Torek operation is so widely used that has caused orchidopexy to fall into such disrepute as it is held by Charny. MacCollum and Gross do not use it; MacCollum used no fixation and Gross used gentle traction with strict injunctions about the pull allowed on the traction. This and their technical skill is likely to have been a major factor in their success rate of 75 to 80 per cent. There is another point of technique which seems to merit consideration. If there is some temporary reaction to manipulation of the testis during the operation it will manifest itself as testicular swelling. This occurs against the resistance of the tunica albuginea and can cause a pressure necrosis. It would seem logical to slit the tunica albuginea at operation. I have done this for several years now and feel that, although its usefulness is still unproved, it does no harm. It certainly seems to lessen the postoperative discomfort of the patient to a marked degree. It also allows a biopsy to be taken.

If orchidopexy is accepted as the method of choice it remains to be decided when it should be done. There are three considerations: (1) the amount of damage incurred by the testis in its abnormal position and the increase of this with time; (2) the influence of the psychologic factor; (3) the controversial possibility of spontaneous descent.

Although more and more biopsy studies are appearing no definite conclusion can be stated yet. The amount of damage seen at orchidopexy at different ages seems to vary (compare fig. 9, age 12½, with fig. 6, age 10). Although it is usually accepted that changes of gradually increasing fibrosis can be seen from 2 years onward this does not seem to be very marked until the first indication of puberty appears, between 8 and 10 years. At that time the amount of peritubular fibrosis increases and tubular maturation lags. Yet, the biopsies shown in the cases represented here suggest either that a high percentage of testes are basically abnormal, or that the damage due to maldescent may often be earlier and more severe than is usually accepted. The unknown factor is the extent to which these changes are reversible. Other than the case of Rea,[39] the best evidence is from MacCollum[33] and Gross.[20] Of the 18 bilateral cases MacCollum reported, one was operated on at 3 and one at 5 years; the rest between 7 and 11 years. Gross states that the majority of his series were operated on between 9 and 11 years. In the light of their good results it seems unnecessary to recommend operation any earlier. On the other hand what are the advantages of waiting this long? Gross states that the operation is too difficult, because of the flimsiness of the structures, before this. Nevertheless, in consideration of the histologic changes and because of the psychologic factor, operation in infancy can be justified.

It is unpopular among clinicians to emphasize the psychologic factors. However, in recent conversations with child and adult psychiatrists, I have been surprised to note the tremendous importance they attach to it. It seems it should be given very much more thought. It is a strong argument to counter Charny's[8] suggestion that there is little justification for treating unilateral cases at all. He considers the results of surgery to be only of cosmetic value in most cases. A single normal and normally descended testis is all that is necessary for fertility. Unfortunately, it is not all that is necessary to satisfy the id. Scorer,[31] who does not think that any spontaneous descent occurs after the end of the first year, advocates a plan whereby the infant is closely observed during the first year. If the testis is palpable at the inguinal ring and does not move during the first year, then orchidopexy should be performed. He claims that it is a reasonably easy operation at this time and that he finds the testis is resting on an abnormal sling of Scarpa's fascia which is attached to the pubis across the entrance to the scrotum. He does not give his recommendations if the testis is not palpable. It seems that his suggestion should be given careful consideration but

that if the testis is not palpable at all it would be better to wait until the infant is older before performing the extensive operation that may be necessary in such cases.

A properly performed orchidopexy is the most satisfactory method of treatment according to the present evidence. It may be argued that it should be done early, certainly before it becomes noticed as a stigma by the patient but there is evidence that postponement until 9 or 11 still gives good spermatogenic results.

4. Orchiectomy. This is advocated because of the danger of the development of neoplasm. In unilateral cases unless the testis can be brought down to the scrotum after two operations it is probably better to remove it. If it remains intra-abdominally it is likely to be late before any neoplastic change can be detected. After puberty, in unilateral cases, it is better to remove the testis than to bring it down. The testis will have no spermatogenic function and orchidopexy will not prevent the development of neoplasm.[19] If the psychologic factor in these cases is as important as the psychiatrists suggest, perhaps a prosthesis should be substituted when orchiectomy is performed. In bilateral cases orchiectomy should not be performed. Such an operation will convert the cryptorchid into a eunuch since even in bilateral abdominal retention the Leydig cells develop and secrete sufficiently to maintain virility.[15]

CONCLUSION

The following method of management is recommended, taking into consideration the present state of knowledge.

The patient should be seen early, preferably at birth, and carefully examined so that the abnormality can be classified.

Those with a suggestion of hypogonadism, delayed bone age and evidence of other endocrine disorders should be treated with hormones.

Those who have pseudocryptorchidism should be discovered by repeated careful examination. The development of the scrotum may raise suspicion in doubtful cases. These should be reassured.

Those with ectopia should have orchidopexy. There seems to be no reason to delay this operation and it should be done when the diagnosis is definite.

Those with incomplete descent should have orchidopexy without a "therapeutic trial" of hormone therapy since this may be damaging to the testis. Although operation might best be done in infancy, particularly if the testis is palpable, the confidence of the surgeon in handling the tissues should be the governing factor. If it is decided not to operate in infancy, the operation should be postponed until 5 or 6 years, when its nature can be explained to the child.

The orchidopexy must be done by a surgeon conversant with the problems likely to be encountered and must be performed with meticulous attention to detail in preserving the blood supply both at operation and afterwards. Otherwise orchidopexy is worse than no treatment at all since it can cause not only a failure of spermatogenesis but atrophy of the Leydig cells as well.

SUMMARY

The factors involved in the normal descent of the testis are discussed.

Some of the reasons for aberration of this process are reviewed.

The five types of maldescent are described: secondary cryptorchidism, primary testicular dysplasia, incomplete descent, ectopia and pseudocryptorchidism.

The effects of maldescent are outlined with particular reference to spermatogenesis and psychologic trauma.

Four methods of treatment are evaluated: watchful waiting, hormone therapy, orchidopexy and orchidectomy.

A plan of management is suggested.

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ACUTE CARDIOPULMONARY EMBARRASSMENT: REPORT OF A CASE*

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Modern surgery has blossomed under the nurture of three significant advances in the past century. These are: the control of pain, the control of infection and an understanding of the mechanisms of shock. There remain, however, difficult problems to be answered with regard to refractory and irreversible shock. An unusual case of cardiogenic shock followed by refractory shock is to be reported.

CASE REPORT

A 20-year-old white man was admitted to the hospital on October 3, 1958, complaining of progressive abdominal swelling. The patient's history was typical of Hirshsprung's disease which had been present since birth. One year before admission an operation for removal of the spastic segment had been performed with incomplete resection of the aganglionic portion of his rectum. After this procedure he did well for a short time and then developed abdominal distention. In the few weeks just before admission, he became rapidly worse with paradoxical diarrhea, severe abdominal cramps, respiratory distress, vomiting, as well as distention. His paternal grandfather had a similar condition.

At the time of admission, the patient was in severe distress. He was apprehensive, tachypneic, pale and having pain which required narcotics. Massive abdominal distention was evident. There were prominent veins over the abdomen. Rectal examination revealed considerable perianal scarring, some voluntary rectal tone and bowel of normal caliber inside the anal canal. X-ray films of the abdomen revealed massively distended loops of large bowel (fig. 1). After admission, the patient's condition quickly deteriorated. Attempts to decompress the abdomen from below were ineffective. Despite this he continued to have small, liquid bowel movements.

Because of the enormity of the anesthesia risk, a cecostomy with local anesthesia was performed. Free, serous, abdominal fluid was encountered during the introduction of a No. 34 French catheter into the cecum. This catheter functioned poorly even though frequently irrigated and the patient's condition continued to deteriorate. His distention became worse and his pulse rate rose to 160. Profuse diaphoresis and cyanosis were present. The blood pressure fell to 98/80 and respirations increased to 54. The electrocardiogram showed sinus tachycardia with a mean QRS vector pointing to 15° in the frontal plane. This axis deviation changed to 60° with slowing of the rate in the postoperative period (fig. 2). X-ray films of the chest revealed extremely high diaphragms, which were elevated to the second anterior interspace on the right, and marked distortion of the heart shadow (fig. 3). The patient was rapidly digitalized. He was irrational and responding only to pain at the time of introduction of anesthesia.

On October 5, at 2 a.m. abdominal exploration and decompression was performed. Only small percentages of cyclopropane were used to accomplish satisfactory anesthesia. A modest upper abdominal incision was made, revealing very large, pulsatile, distended veins in the midline. The peri-

FIG. 1. Flat film of abdomen revealing massive colonic distention. Cecum measured 23 cm. in diameter in this projection on the original film. Rayl tube is in the stomach.

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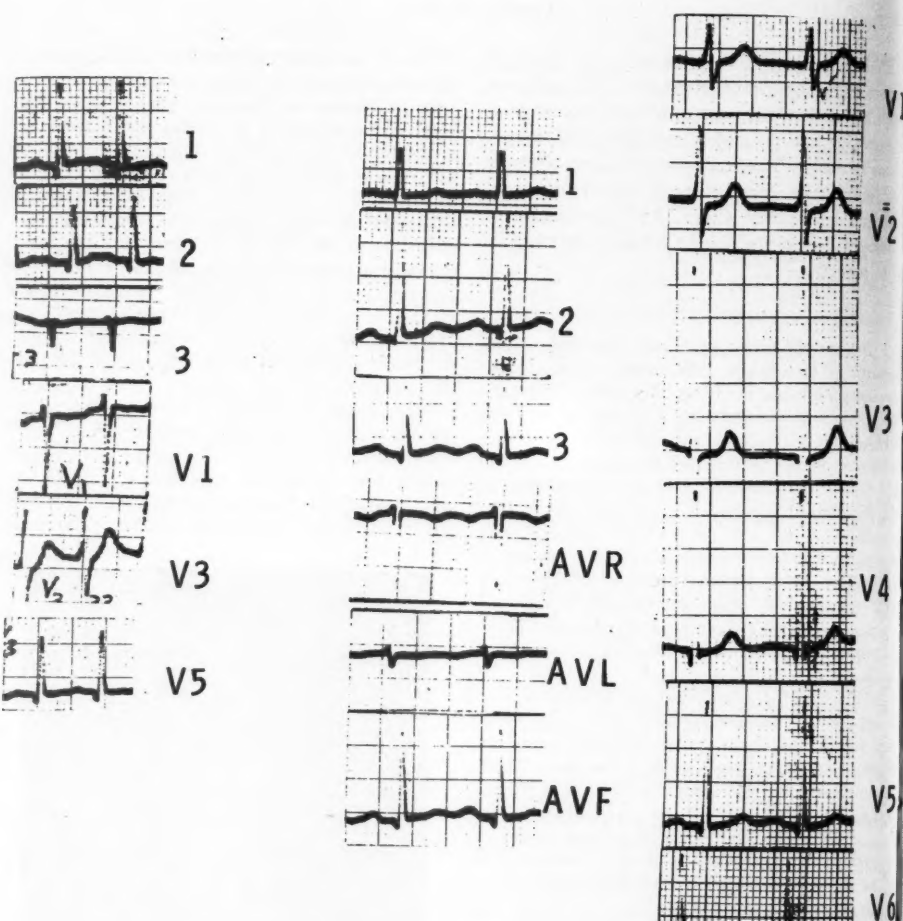

FIG. 2. Electrocardiograms taken before decompression and digitalization and in the convalescent phase. Note the axis deviation and the tachycardia.

toneum was opened with caution and clear, serous fluid filled the cavity. A knuckle of tremendously dilated and thickened colon was then opened and its contents aspirated. Subsequently, the wound was enlarged and multiple colotomies were performed with several thousand milliliters of semisolid feces and gas being aspirated. There was an immediate rise of blood pressure to 120/70 and a fall in the pulse and respiratory rates. The patient's color improved. Soluble Neomycin, ½ gm., was instilled in the abdominal cavity in 100 ml. of normal saline solution before closure of the wound around a knuckle colostomy. The patient then tolerated being placed in a lithotomy position. A small posterior midline incision was made in the anal canal and after dilation a large tube with the

FIG. 3. Chest x-ray taken just before surgery. There is marked distortion of the cardiac shadow and extremely high diaphragms. Although posterior lung vascular markings are seen, the liver casts only the faintest shadow. No splenic shadow can be made out.

caliber of a garden hose was introduced toward the hollow of the sacrum. Another gush of fecal material was encountered after this tube passed about 12 cm. into the colon. An estimated 8000 ml. of gas and a measured 5000 ml. of liquid feces was removed from the bowel by aspiration in the operating room.

Postoperatively, the patient's blood pressure gradually fell to shock levels again with several readings around 60/40 being recorded. With a slow intravenous drip of norepinephrine and abdominal sand bags the hypotension was controlled. Because of a fear of peritonitis and a positive culture on the abdominal fluid of a coliform bacillus, adrenal steroid therapy was not begun until October 8, despite the pleadings of our medical colleagues. About 24 hours after hydrocortisone (200 mg.) was given, cardiovascular balance became re-established. Antibiotics were continued for 1 week.

Four subsequent minor operative procedures have proved necessary, three of which were performed under local anesthesia. With progressive decompression from the cecostomy and rectal tubes, the knuckle colostomy was taken down and a secondary closure performed on October 10th. It soon became apparent that long term decompression would be necessary for the bowel to return to a more normal caliber. Therefore, a divided-loop transverse colostomy was performed on November 14. Before the patient's discharge, a colostomy revision and an operative repair of the cecostomy were done. Biopsy at the site of the colostomy revealed normal numbers of ganglion cells. Postoperative studies have included a barium enema, an intravenous pyelogram and another electro-

FIG. 4. Barium enema indicating length of remaining spastic segment.

FIG. 5. Intravenous pyelogram showing minimal ectasia of the right renal pelvis.

cardiogram (fig. 2). The colon contrast study was done before the colostomy and revealed a spastic segment of the anticipated length (fig. 4). Intravenous pyelogram was interpreted as showing some minimal dilation of the right renal pelvis (fig. 5).

Future plans for this patient include an attempt to completely excise the aganglionic segment and re-establish the normal bowel continuity. This must await a prolonged period of decompression. At this time, he is being followed as an outpatient with colostomy care and anal sphincter exercises.

DISCUSSION

James Latta first introduced to medicine the word "shock" in its present day connotation (as quoted by Parsons and Phemister[9]) in 1795. The subsequent history of medical thought and endeavor in the area of cardiovascular collapse is presented in some of the classic papers on this subject.[1-3, 5, 7-10] As suggested by the word itself, early investigators relied on Claude Bernard's demonstration of the sympathetic control of blood vessels for their theories. The proferred mechanism by which severe mental or nervous disturbances caused shock was by way of these sympathetic controls. When the role of peptones and histamines in the production of shock became known, a new school of thought was formulated. This school found strong support in such personages as Heidenhain, Dale, Cannon, Bayliss, Sherrington, Starling and Sir Thomas Lewis.

Our current knowledge with regard to initiating and precipitating factors in shock is well founded on the beautifully documented and controlled experiments of Doctors Blalock and Phemister. The conclusions from these studies stand out in sharp contrast to the conclusions of an earlier classic work by the British physiologists Cannon and Bayliss, where similar techniques were used.

If one excludes myocardial infarction, acute circulatory failure as a result of primary disturbance of the heart is a rare occurrence. It differs, of course, from hematogenic, neurogenic, and vasogenic shock in that venous distention is present. In this unusual case, there seems little doubt that the preoperative shock was on a mechanical basis. The extreme displacement of the heart and lungs, coupled with the marked compression of lung tissue and mediastinal contents, are undoubtedly responsible for the lack of adequate circulation and ventilation. Intrathoracic pressure relationships were shifted markedly in the positive direction. Inflow impedance to venous blood was high resulting in diminished cardiac filling and output. The desaturation of arterial blood from embarrassed ventilation and pulmonary perfusion compounded the problem. Similar interesting but not identical mechanical problems are seen in constrictive pericarditis, mediastinal shifts, cardiac tamponade, paroxysmal arrhythmias and pulmonary embolism.

The refractory shock seen in the postoperative period is less easily understood. It seems quite likely that both neurogenic and vasogenic factors were at play. The delayed response to hydrocortisone would seem to cast some doubt on adrenal insufficiency as a major perpetrating factor; however, the adrenals may well have had a role, acting *via* antagonism of a blood-borne factor. There is established laboratory evidence which seems to show that the gluco-corticoids antagonize histamine release from traumatized cells or other areas.[4] Furthermore, recent experimental evidence suggests that promethazine (Phenergan) and deoxycorticosterone acetate protects against postadrenalectomy shock, whereas pretreatment with deoxycorticosterone acetate alone is unsuccessful.[6] Liver, bowel and renal ischemia can produce "toxic factors" causing irreversible shock. Artificial perfusion with oxygenated blood and antibiotics may protect the experimental animal from this phenomenon. Pooling of blood in the splanchnic bed may also be a factor here as it is sometimes seen following paracentesis. These are the areas where most modern research efforts are deployed, and where most of the present day riddles with regard to shock are to be found.

SUMMARY

A case of cardiogenic shock from extreme displacement and compression of thoracic structures has been reported.

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ADDENDUM

Since this article was submitted for publication the patient has had his definitive surgery. On April 17, 1959, a Swenson type of pull-through and resection of his distal colon was performed without incident. The aganglionic segment was of the anticipated length.

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AN UNUSUAL COMPLICATION OF MECKEL'S DIVERTICULUM

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INTRODUCTION

The recent occurrence on our surgical service of an unusual complication associated with a Meckel's diverticulum in a 9-year-old male child prompted us to review the literature concerning this entity. The peculiarities of the clinical situation and pathologic anatomy discussed here have not been previously described to the best of our information. The problem presented to us was that of a Meckel's diverticulum with a subfascial anterior abdominal wall dissecting abscess and with a confusing and alarming clinical picture.

The literature is replete with the ordinary complications that one may encounter while dealing with the problem of a Meckel's diverticulum. Penberthy[7] lists as usual complications: umbilical discharge, cystic growths at the umbilicus, inflammation, perforation, acute hemorrhage, intussusception and obstruction. Morlock and Bennett[6] write of additional complications of umbilical fecal fistula, calculus formation in the Meckel's diverticulum and neoplastic changes in the mucosa. Mohardt[5] describes the occurrence of carcinoids in Meckel's diverticula. Prolapse of the ileum through an umbilical fecal fistula *via* a Meckel's diverticulum is reported by Horsley.[2] A rare case with a twisted Meckel's diverticulum with infarction and production of severe pain, rigidity and simulated peritonitis is described by Ladd and Gross.[3] Heterotopia is commonly reported in Meckel's diverticula and mentioned as perhaps being instrumental in the existence of some of the above listed complications.[4] The discussion of historic background, embryology and anatomy, and the clinical picture with discussion of incidence, symptoms, diagnosis and treatment are amply covered in the literature and will not be discussed here.

CASE REPORT

R. B., a 7-year-old white boy, was referred because of severe abdominal pain, fever and signs of what was thought to be a generalized peritonitis of 12 hours' duration.

Present illness. The recent illness began about 36 hours before being seen by us when the patient suddenly developed severe abdominal pain, became nauseated and vomited once. He was taken to his family physician who noted mild fever, minimal periumbilical tenderness and a white blood count within normal limits. He was given an injection of a penicillin-streptomycin mixture and was sent home to return the following day. When seen 12 hours later, the patient was acutely ill, his fever was 102°, and he had marked abdominal pain on palpation. A diagnosis of peritonitis was made and the patient was transferred to our hospital.

History. Of significance in the development of this history is the fact that this child had suffered repeated attacks of cramping, periumbilical abdominal pain for 5 years or more. The attacks were extremely variable in their severity, duration and periods of remission. There was no other notable or pertinent factor in the past history; his development was normal, his habits were usual and he had experienced no major illnesses. The child has a second cousin who was operated upon by us in 1956, at age 26, because of chronic gastrointestinal bleeding of undetermined etiology. This man was found to have had a Meckel's diverticulum containing gastric mucosa and an associated ileal ulcer.

Physical examination. On admission the physical findings were as follows: rectal temperature, 104°; pulse, 130; respirations, 44. The child was flushed, apprehensive, dehydrated, in obvious pain and acutely ill. Small lymph nodes were palpable in the cervical and axillary regions bilaterally. The sclerae were injected. Both eardrums appeared normal, the nose was clear and the throat was not remarkable. Chest examination showed rapid, shallow respirations and fine basilar rales were heard bilaterally without evidence of consolidation. The abdomen was slightly distended. The umbilicus appeared normal; bowel sounds were diminished to absent. There was severe muscle spasm and production of great pain to even the slightest pressure in the entire lower abdomen from the umbilicus caudally, the upper abdomen illiciting pain and spasm on palpation but not to as great a degree as in the lower abdomen. There was no rebound localization. Rectal tenderness was absent. There was no evidence of inguinal hernia. The testicles and penis were normal. Costovertebral angle tenderness was absent. Peripheral pulses were intact. The extremities were not

remarkable. The neurologic examination was within normal limits.

Laboratory reports. On admission the white blood cell count was 14,600 with 88 P.M.N.'s, 5 stabs, 6 lymphocytes, 1 monocyte; hemoglobin, 11.5 gm.; red blood cell count, 4,100,000. Urinalysis: sp. gr., 1.032; albumin, trace; color, clear; microscopic, negative for red blood cell counts, white blood cell counts or casts.

X-ray reports. On admission, flat and upright films of the abdomen showed no free air, but were consistent with an ileus with some distention of the small bowel and a greater distention of the colon. Chest x-rays were interpreted as normal other than slightly increased density due to decreased respiratory excursion.

Course in hospital. After consultation with a pediatrician and an anesthesiologist, it was agreed that operative care should be delayed until the patient's immediate status could be improved. He was placed on a penicillin-streptomycin mixture intramuscularly, and was given 500 cc. of 5 per cent dextrose in water intravenously. He was given 25 mg. of Demerol for pain and an alcohol bath and aspirin per rectum to lower his temperature. Several hours later when his temperature reached 100° (rectal) and his respiratory rate lessened, he was given preoperative medication. Anesthetic agents used were sodium Pentothal, nitrous oxide and curare.

Operative procedure and findings. A right rectus incision was made. After dissecting through the anterior rectus sheath, pus was encountered in the periumbilic area. It was found that the purulent

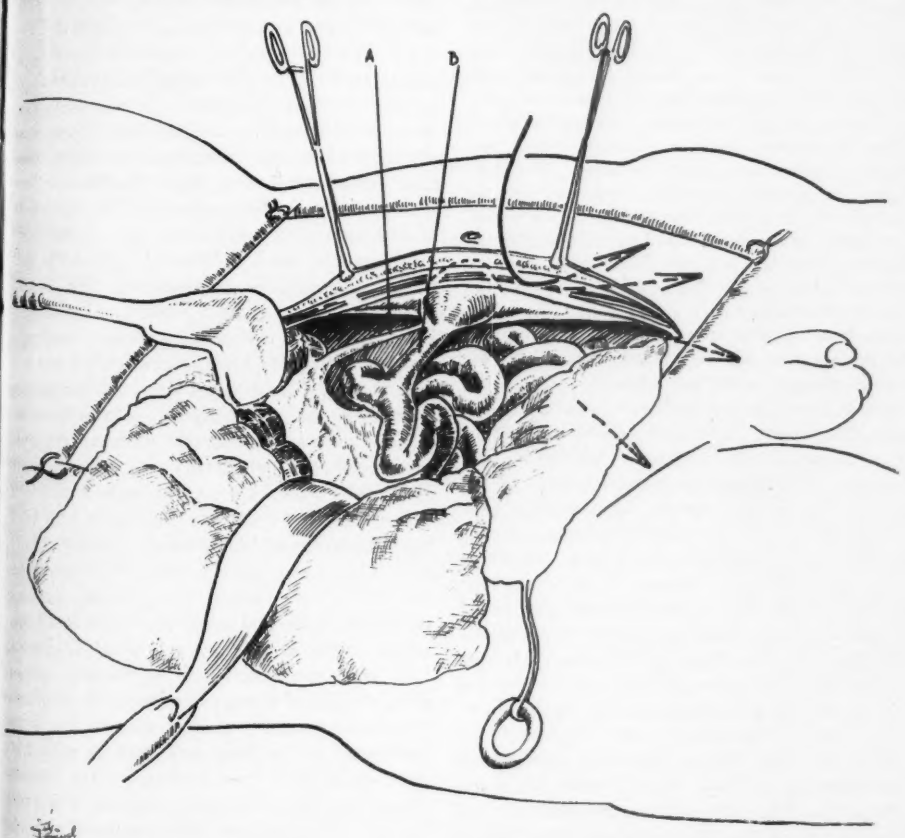

FIG. 1. Illustration showing findings encountered after peritoneal cavity was opened and after the small bowel was derotated. *A*. Vascular arch. *B*. Meckel's diverticulum with cystic enlargement at its distal end. Arrows illustrate path of dissection of pus beneath anterior rectus sheath and into both inguinal areas.

material lay immediately beneath the anterior rectus sheath on both sides of the midline. It had dissected beneath this layer in a fanlike form to both inguinal regions (fig. 1). Cultures were obtained. The purulent material was gently cleansed away and the peritoneal cavity was entered. There was minimal free fluid in the peritoneal cavity, without evidence of a frank peritonitis. A large caliber persistent omphalomesenteric duct was found extending from the terminal ileum to the umbilicus and a small perforation was found in the cystlike enlargement at the distal end of the duct. The perforation was open into the space between the anterior and posterior rectus sheaths at the midline. Lying immediately adjacent to the ophalomesenteric duct and existing as a separate structure was an anomalous vascular arch with a diameter the size of a small lead pencil. It arose from the root of the mesentery and extended to the umbilical area paralleling the omphalomesenteric duct. Thus, there were created two pillars, one from the ileum and one from the mesenteric root, and the small gut was rotated 360 degrees about these structures producing an incomplete mechanical obstruction. After derotation of the small bowel, the omphalomesenteric duct was clamped and divided in a transverse fashion at the ileum. The defect in the small bowel was closed, and the duct and vascular arch were then excised from the anterior abdominal wall, resecting with them the cystic enlargement at the end of the duct. The wound was closed in layers with fine wire sutures. Large caliber drains were placed beneath the external oblique fascia and allowed to come out through separate stab incisions in each inguinal area. Drains were also placed between the anterior and posterior rectus sheaths bilaterally and brought out through the inferior margin of the wound. The patient tolerated the procedure well.

Postoperatively, he was given intramuscular injections of a penicillin-streptomycin mixture twice a day and 250 mg. of Terramycin intravenously, twice a day. On the 2nd postoperative day, bowel sounds were normal and the patient began to eat. With the exception of a minor pulmonary infection on the 3rd and 4th postoperative days, his response was gratifying. Pathologic diagnosis of the operative specimen was that of a persistent omphalomesenteric duct. The vascular arch showed only the presence of blood vessels, nerve fibers and lymphatics. The cultures of the purulent material showed no growth. His drains were removed on the 8th postoperative day and the patient was discharged from the hospital. Follow-up examinations of the patient have shown complete recovery.

COMMENT

There are many interesting facets of this clinical picture as presented in the case report. Foremost of these, of course, is that of the persistent omphalomesenteric duct with the free perforation occurring not through the skin to the outside thus forming a fecal fistula, nor a free perforation into the peritoneal cavity producing a peritonitis, but a free perforation occurring between the anterior and posterior rectus sheaths of the abdominal wall thus creating the unusual sequence of events. The sudden onset of pain in this child must have been occasioned by the occurrence of the perforation. The relative early nontoxic response as observed by his family physician was not alarming. As the fecal contents continued to pour through this perforation and as suppuration developed and began to dissect through the layers of the anterior abdominal wall, the child became more ill and the picture became more confused. Of extreme significance, we believe, is the fact that this child showed exquisite tenderness throughout the entire lower abdomen at every point caudal to the umbilicus with even the lightest palpation. The expression of such agonizing pain to such light pressure almost made us think in terms of a psychotic episode. It is evident in reconstructing this clinical description that even light pressure to the acutely inflamed anterior abdominal wall must have produced terrible pain. Also of significance is the fact that there was no external sign of inflammatory change such as one usually associates with an infectious process. Eisendrath[1] discusses six anatomic possibilities which may occur with Meckel's diverticula and which may be present at birth. These are: (1) a complete canal extending from the ileum to the umbilicus which is patent at both ends; (2) a canal which is open at the umbilicus but which ends blindly at a variable distance within the abdominal cavity and which is not connected to the ileum; (3) a cyst which is blind at both ends and which may be variably placed along the line of the usual course of the omphalomesenteric duct; (4) a canal open at the ileum, but closed at its distal end with or without a stalk attached to the umbilicus; (5) a vascular cord which may be variable in position, which may be free or attached, and which is usually termed the "terminal ligament" when it extends from a Meckel's diverticulum to the umbilicus; and (6) a remnant of vessels from the mesentery to the

umbilicus which may exist as a cordlike structure. The pathologic anatomy here was that of a canal open at the ileum extending all the way to the umbilicus but not open at the umbilicus (fig. 1). In addition there was a vascular arch from the free edge of the mesentery to the umbilicus.

SUMMARY

A case of an unusual complication of Meckel's diverticulum in a 9-year-old white male is presented. This complication is that of a perforation of a complete Meckel's diverticulum into the fascial planes of the anterior abdominal wall producing an unusual and interesting clinical picture. We believe this to be a previously undescribed complication occurring in a Meckel's diverticulum.

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ARTHRODESIS OF THE PARALYTIC SHOULDER*

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Key[2] made the following statement: "Arthrodesis of the shoulder is an operation which I have recommended and performed on rare occasions. This is partly because the operation is relatively difficult, and, in my hands, rather unsatisfactory in that I know of no operation which fulfills the requirements for a satisfactory arthrodesing operation. These are the bringing of sufficiently large areas of cancellous bone together and holding them in firm contact until bony union has occurred, or the extra-articular fixation of the joint by adequate extra-articular grafts which will proliferate and afford a strong union between the bones." I believe the operation to be described overcomes these difficulties.

The operation to be described is not entirely new. Brett[1] in 1933 described a similar procedure and reported two cases. Roundtree[4] should be credited with the following method for fusion of the shoulder joint, since it was devised by him and he has used it for many years; although, it has not been previously reported. In a personal communication, he told me that he had done the operation approximately 45 times and that he was very gratified with his results.

SURGICAL TECHNIQUE

Preoperatively, the patient is placed in a shoulder spica. The ideal position recommended by the Research Committee of the American Orthopaedic Association[3] is 45 to 55 degrees of abduction, 15 to 25 degrees of flexion and 15 to 25 degrees of internal rotation. Just before surgery, the cast is bi-valved and the upper shoulder and arm portion removed. With the patient in the remaining portion of the cast, the extremity can be easily draped. A longitudinal incision is made from the acromial process downward onto the shaft of the humerus for a distance of approximately 3 inches and the shoulder exposed. The head of the humerus can easily be delivered into the wound and denuded of its cartilage. It is then displaced and the glenoid is likewise denuded of cartilage. Next, a hole is drilled at approximately a 45-degree angle, starting in the shaft and drilling toward the center of the head. The size of this drill hole will vary with the size of the patient, but, it is usually approximately ¼ inch in size. The humerus is placed in the desired position in contact with the glenoid and the drill hole is extended into the glenoid fossa and the neck of the glenoid. A peg of cortical bone, slightly larger in diameter than the drill hole, is fashioned with a pointed end. This is driven through the humerus into the glenoid and any excess of bone that may be protruding from the humerus is removed with a rongeur. The wound is closed in the usual manner and the bi-valved portion of the cast is reapplied.

FIG. 1. Case 2. Photograph made 6 months after surgery shows typical result.

SUMMARY

Arthrodesis of the paralytic shoulder is usually reserved for those patients having a fair to good trapezius and serratus anterior; however, it is feasible to fuse the completely flail shoulder, since it gives the patient an added sense of security, facilitates dressing and undressing, makes it easier to turn in bed and allows the patient to place the extremity on a table to brace his paper for writing. Time of immobilization required for fusion is usually three months. Premature closure

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FIG. 2. Case 6. Radiograph made 3 years following fusion. The epiphysis is still open

TABLE I

Data on six patients with shoulder fusion

Case	Age at Onset	Age at Surgery	Fusion Obtained	Muscles	Result
1	2 yr.	8	3	Good trapezius; good serratus anterior; good hand, elbow and wrist.	Excellent
2	6 yr.	10	6	Good trapezius; good serratus anterior; good hand, elbow and wrist.	Excellent
3	7 mo.	6	4	Good trapezius; good serratus anterior; good hand, elbow and wrist.	Excellent
4	6 yr.	14	3	Good trapezius and serratus anterior; good hand and wrist; no elbow flexor (Stienler flexoplasty).	Good
5	3 yr.	10	6	Good trapezius; good serratus anterior; good hand, elbow and wrist.	Excellent
6	2 yr.	10	4	Good trapezius and serratus anterior; no forearm supination.	Good

of the humeral epiphyseal plate has not been noted. Bone bank bone was utilized in all six cases of this series (table 1). We have described a simple and effective means of obtaining an arthrodesis of the shoulder which I feel deserves a wider use.

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RETROGRADE JEJUNOGASTRIC INTUSSUSCEPTION: REPORT OF A CASE FOLLOWING SUBTOTAL GASTRIC RESECTION*

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Intussusception of one part of the intestinal tract into another part has long been recognized as a definite disease entity. Hunter[7] described intestinal intussusception in 1793, and since that time many papers on the subject have been presented. Three types of intussusception have been described. The most common is the ileocolic which involves both the terminal ileum and the colon; the next most common is the enteric which involves only the small intestine; and the least common is the colic which involves only the colon. A fourth type, involving the stomach and jejunum, will be discussed here, and a case of jejunogastric intussusception following a subtotal gastric resection will be presented.

The ileocolic, enteric and colic types of intussusception almost always occur in an antegrade manner, although they may occur in retrograde.[12] It has been reported that this ratio is 200 to 1.[1-5] The jejunogastric type following simple gastroenterostomy may occur with either the afferent limb, the efferent limb, or both afferent and efferent limbs invaginating into the stomach.[4-11] The only type following subtotal gastrectomy, as revealed by a review of the literature by Prohaska and co-workers,[10] is the retrograde invagination of the jejunum into the remaining gastric pouch.

Jejunogastric intussusception following gastroenterostomy was recorded first in 1914.[2] In 1957 Prohaska and co-workers[10] reviewed the literature and found that 81 patients had been reported with this complication following surgery. Of these patients, 66 had simple gastroenterostomies and 15 patients with jejunogastric intussusception had subtotal gastric resections.

Osmond and Fowler,[9] found that a few more than 100 patients with jejunogastric intussusception had been reported in the world literature. They did not divide these patients according to the type procedure previously performed.

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DIAGNOSIS

In arriving at the diagnosis of jejunogastric intussusception, a careful history and good roentgenograms of the stomach are most important. The physical examination and laboratory data are not helpful in making a diagnosis in most patients. The time interval between gastric resection and the occurrence of jejunogastric intussusception varies widely. The shortest interval recorded was 11 days, and the longest was 20 years.[10] The condition may be of the severe acute type and require immediate treatment, or it may be of the chronic recurrent type and continue to recur for years before treatment is actually required.[3]

Early diagnosis and proper surgery are extremely important in those patients with the severe acute type of jejunogastric intussusception. The mortality rate is as high as 10 per cent in those patients operated on within 48 hours after the onset of symptoms, and rises to 35 per cent to 50 per cent when surgery is delayed.[8] The most common findings noted in the acute type of jejunogastric intussusception are as follows.

History: (1) In previous gastric surgery in which the jejunum has been anastomosed to the stomach. (2) After a varying interval of no symptoms, there is a sudden onset of severe cramping, epigastric pain, and vomiting of food and bile, and later, blood-tinged fluid.

Physical: (1) The abdomen may be tender, and in some patients there may be abdominal rigidity, but these signs are not constant. (2) A mass may be palpated in about 50 per cent of the patients.[6]

X-ray: (1) Roentgenograms of the abdomen are significant when a clinical picture of small bowel obstruction is present and no dilated loops of small bowel are demonstrated. (2) Fluoroscopic examination reveals a movable mass within the stomach having curved lines simulating the pattern of intestinal folds. (3) There may be delayed gastric emptying. (4) The stomach is dilated.

CASE REPORT

Z. G., a 67-year-old colored man was admitted to Nashville General Hospital April 2, 1958, with the chief complaint of abdominal pain. His history revealed that for some time he had had episodes of "stomach ache" not relieved by food and often made worse by eating. He had been nauseated a few times and vomited, but no blood had been noticed in either vomitus or stools. Three days before admission he developed severe, mid-epigastric cramping pain which radiated to the umbilicus. Two days later he became nauseated and vomited "two jars" of blood. The past history revealed that in 1947 he had had a subtotal gastric resection for duodenal obstruction.

On physical examination the blood pressure was 150 systolic over 110 diastolic; pulse, 110 per minute; temperature, 99°; and respiration, 18 per minute. The patient was a well developed, fairly well nourished man. The only significant physical finding was marked dehydration. There was no palpable abdominal mass and no blood in the stool. The clinical impression on admission was upper gastrointestinal bleeding, possibly due to a marginal ulcer, carcinoma of the stomach, or esophageal varices. The hemoglobin was 16.2 gm. with a packed cell volume of 54 per cent. A Levin tube was passed into the stomach and the patient was found to be actively bleeding. He was given intravenous Premarin, whole blood, glucose and intramuscular Adrenosem, but the active bleeding continued. Approximately 30 hours after admission, 4 days after onset of the present illness, an exploratory laparotomy was done.

The anesthesia was sodium Pentothal and gas, oxygen and ether and Anectine for skeletal muscle relaxation. The abdomen was entered through an incision made obliquely from the left costal margin downward and to the right, and extending distally just to the right of the midline to the level of the umbilicus. Exploration revealed a previous gastric resection with a Pólya type anastomosis and a markedly dilated stomach. The stomach was opened, and 2 feet of gangrenous intussuscepted jejunum was found in its lumen (figs. 1 and 2). It was advisable not to reduce the intussusception owing to the edema and gangrene of the intussusceptum. The intussusception was resected including the distal few centimeters of the stomach and the gangrenous portion of the jejunum. The continuity of the jejunum was reestablished by an end-to-end anastomosis. A Hofmeister antecolic gastrojejunostomy was made between the open end of the stomach and a more distal segment of the jejunum. The patient's condition was poor from the beginning of the operation, and the blood

FIG. 1. Gangrenous loop of jejunum intussuscepted through stoma into stomach.

FIG. 2. Specimen following excision

pressure was maintained at just above shock level throughout the procedure. The abdomen was closed in layers, and he was returned to the ward in poor condition.

Approximately 4 hours after surgery the blood pressure fell to 50 systolic over 40 diastolic. A subnormal blood pressure persisted despite vasopressor drugs for most of the remaining hospital stay until his death on the 3rd postoperative day. An autopsy was not obtained.

SUMMARY

Retrograde jejunogastric intussusception has been discussed, and a case following subtotal gastric resection has been presented. The most common findings in the history, physical and roentgenogram examinations have been reviewed. Early diagnosis and proper surgery are of the utmost importance for the survival of the patient.

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TRICHOBEZOAR: REPORT OF A CASE COMPLICATED BY MULTIPLE GASTRIC POLYPS*

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Trichobezoar may be defined as a foreign body in the stomach or intestinal tract which consists of hair, undigested food particles and mucus. Baudmant, in 1779, recorded the first authentic case of trichobezoar.[1-7]

Most of the reported cases of trichobezoar have been in young women. In the majority of the patients it is not clear why the perverted habit of hair-eating exists. Only a few of the patients have been insane; however, most of them have had neurotic tendencies. Butterworth[2] suggests that the custom of young girls wearing their hair long is an important factor in favoring the practice of biting the hair or passing the strands between the teeth.

There has been a total of 208 patients with trichobezoar reported in the literature, only one of which was associated with gastric polyps.[3] This is the second reported case of a patient with both gastric polyps and a gastric trichobezoar.

Most trichobezoars are located in the stomach; however, a few have been reported in the small intestine.[6] They have varied in weight from 2¼ ounces to 6 pounds before desiccation.[4] In most patients trichobezoars are single, but in a few patients they have been multiple. The general contour of a trichobezoar of the stomach when fully developed, corresponds to the shape of the letter "J."

Large masses of hair often remain in the stomach for long intervals without any marked symptoms or significant interference with health.[10] The stomach has a remarkable tolerance to foreign bodies.

Stelzner,[11] in 1896, is believed to be the first to record a correct preoperative diagnosis of trichobezoar. The most common symptoms are anorexia, epigastric fullness, epigastric pain, headache, nausea, vomiting and weight loss. Frequent signs are malnutrition with pallor and a tender mobile mass in the upper abdomen. Routine laboratory work usually reveals a mild anemia and a mild leukocytosis. Examination of the stool may reveal occult blood. In a few patients hair fragments have been identified in the stool. When the diagnosis is suspected gastric lavage may confirm the diagnosis.[4]

The use of x-ray with contrast media has done much to make the diagnosis of trichobezoar possible. According to DeBakey and Ochsner[5] the first correct roentgen diagnosis of a trichobezoar in the stomach was made by Kaufman in 1911. With a barium meal the trichobezoar produces an irregular negative shadow which can often be seen to move within the barium-filled stomach. Peristalsis is usually absent and the gastric air bubble is frequently obliterated. The 6-hour film shows the barium caught in the interstices along the surface of the trichobezoar.[9] As an added diagnostic procedure, Moersch and Walters[8] have advocated direct visualization by means of gastroscopy. In the differential diagnosis an enlarged spleen, enlarged or mobile kidney, cysts of the liver and tumors of the omentum, pancreas, stomach and colon must be considered.[9]

The prognosis depends primarily upon the presence or absence of two factors: proper diagnosis with the institution of adequate therapy and complications which may arise if the diagnosis is not made. The complications resulting from trichobezoar are due to interference with the normal physiologic activity of the gastrointestinal tract, namely, obstruction and traumatic irritation to the stomach and bowel wall resulting in mucosal erosion, ulceration, perforation and peritonitis.

Therapy consists of laparotomy, anterior gastrostomy and removal of the trichobezoar, care being taken to avoid contamination of the peritoneal cavity with drippings from the putrid mass.

There have been two patients with trichobezoar recorded at Nashville General Hospital. Only one of these patients had associated gastric polyps.

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FIG. 1. Roentgenogram of the stomach with barium which demonstrates a large foreign body diagnosed as a bezoar.

FIG. 2. Gross specimen of gastric trichobezoar removed at surgery.

CASE REPORT

H. S., a 20-year-old white woman, was admitted to Nashville General Hospital on August 13, 1956, with the chief complaint of a mass in the upper abdomen of 5 months' duration. The mass had increased in size since it was first noticed. There had been some dull epigastric pain for the previous 4 months and a weight loss of 3 pounds. For 3 months she had a sensation of fullness and the inability to eat a full meal. There was no history of hematemesis or melena. The remainder of the history was noncontributory.

The physical examination revealed a thin, poorly nourished white woman in no distress. The only positive physical findings were carious teeth and a mobile tender mass in the epigastrium.

Gastric lavage was done but no hair was demonstrated. Gastric analysis revealed no free hydrochloric acid and only three degrees of total acid. Examination of the stool was negative for blood and hair. Routine blood work revealed a packed cell volume of 39 per cent, hemoglobin of 11.83 gm. per cent and white blood count of 7500. The urine was normal. The total serum protein was only 5.5 gm. per cent with a normal A/G ratio. An upper gastrointestinal series was reported as follows: "The esophagus is normal. The stomach is completely filled with what appears to be a large bezoar. Insufficient barium passes for visualization

FIG. 3. Roentgenogram of the stomach 8 months after gastrostomy and removal of trichobezoar. Note deformity in pyloric area due to gastric polyps.

FIG. 4. A section through a polyp of intermediate size showing a sharp line of demarcation between the glands of the polyp and the muscularis of the gastric wall; H & E × 100.

of the duodenum. The 5-hour film shows the stomach to be outlined with flecks of barium which surround the intraluminal mass" (fig. 1).

On August 17, 1956, gastrotomy was performed and a 2-pound trichobezoar was removed (fig. 2). Multiple polyps were noted in the distal half of the stomach. Gastric resection was postponed because of persistent hypotension. She made an uneventful recovery and was discharged from the hospital.

She did well except for a persistent poor appetite. The polyps noted at surgery were still present roentgenographically on April 22, 1957, 8 months after the initial operation (fig. 3). A subtotal gastric resection was done on April 26, 1957, with removal of 75 per cent of the stomach. Her postoperative course was uneventful. Soft movable polyps ranging in size from 0.5 to 2.5 cm. were seen grossly in the lower half of the specimen. Histologically these proved to be benign (figs. 4 and 5).

Her last clinic visit was on April 25, 1958, at which time she was in her 3rd month of pregnancy and had not regained her normal weight. There was no complaint with reference to the stomach. The appetite was good and she felt well.

SUMMARY

A discussion of trichobezoar is presented with the report of an interesting patient with a 2-pound gastric trichobezoar. This is the second report in the literature of a patient with a gastric trichobezoar and associated gastric polyps.

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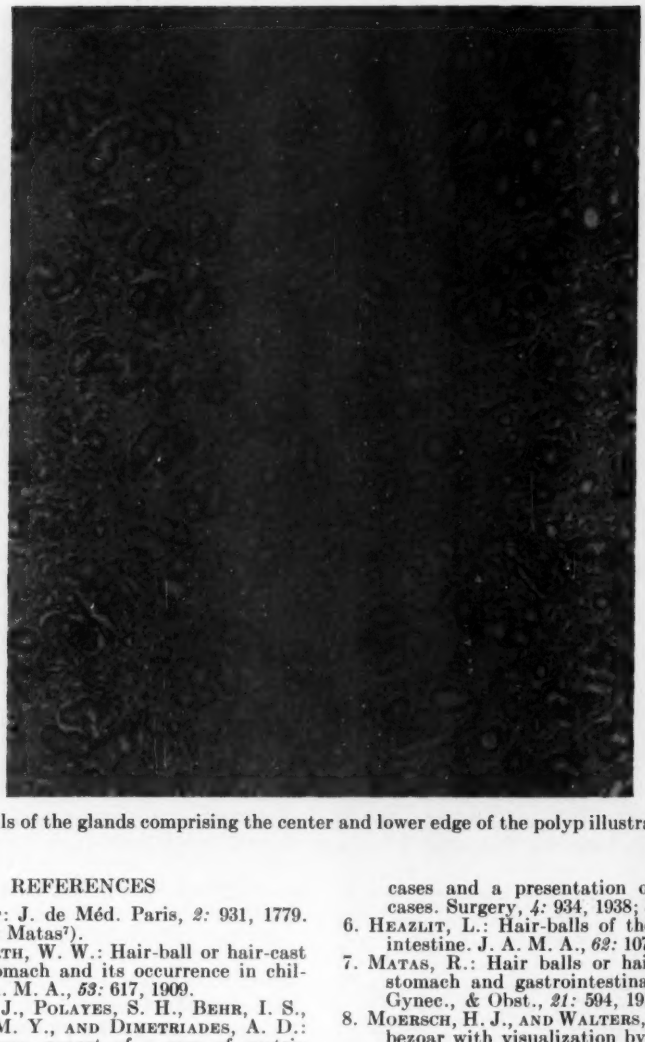

FIG. 5. Details of the glands comprising the center and lower edge of the polyp illustrated in figure H & E × 200.

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Editorial

THE PURPOSE OF A HOSPITAL: GOOD PATIENT CARE OR HOUSE OFFICER TRAINING?

In the past years, changing philosophy and socioeconomic conditions have affected hospitals, postgraduate training and patient care. Formerly, personal philanthropy in the form of monetary contributions permitted the building of hospitals and the care of indigent patients in these hospitals. Taxation, insurance programs, and federal, state, city, and business or union welfare programs have changed these conditions. Today there are few charity patients requiring medical care and few private hospitals that feel an obligation to render free medical service to patients.

When there were fewer hospitals and many indigent patients needing professional medical care in the hospitals, care was given freely, without charge, by both the visiting doctor and the hospital. Since these indigent patients required professional care, it was only natural that there should develop a method of utilizing the talents of recent medical school graduates to care for such patients under supervision of the experienced visiting doctor. It soon became evident under this system that the recent medical school graduate became a better doctor. The recent medical school graduate gradually began to spend longer periods of time in the hospital under the supervision of a competent visiting staff of doctors. Any successful arrangement of cooperative human endeavor must be mutually beneficial. Accordingly, the recent medical school graduate started to help the visiting doctor care for his private patients and this plan was some recompense for the time which the visiting doctor spent in teaching and aiding in the care of the indigent patient. The recent medical school graduate became known as a house officer or house doctor. If he remained in the hospital more than a year he usually began to study a special field of medical work such as medicine, surgery, pathology or some other specialty. This first year became known as an internship. The subsequent years in special training were known as residency training. In Baltimore the graded residency training program was highly developed and resulted in training many of the outstanding professional men in this country. This method of residency training was for the most part adopted by the Advisory Board for Medical Specialties and has become the accepted method of specialty training throughout the country.

The medical schools and various states that were always seeking a higher standard of medical care soon insisted upon an internship. Pennsylvania, in 1914, was the first state to require one year of internship before the recent medical school graduate could obtain a license. The University of Minnesota, in 1915, was the first school to require its medical graduates to serve an internship before receiving the degree of doctor of medicine. In the early years there were not enough internships available for the recent medical school graduates. However, as internships became more abundant they were classified and approved in order to assure adequate training for the intern. The Council on Medical Education and Hospitals of the American Medical Association undertook its first survey of hospitals engaged in intern training in 1912 and published its first list of approved internships in 1914. In the United States, in 1920, there were 2960 approved internships available and 3047 medical school graduates. In 1930 there were 5490 approved internships and 4565 medical school graduates; in 1940 there were 8097 approved internships and 5097 medical school graduates; and in 1957 there were 12,404 approved internships and 6796 medical school graduates.

The method of residency training for the various medical specialties became increasingly popular. In 1914 there were 95 hospitals offering training in some specialized field of medicine. Each practitioner of medicine was the sole judge of his preparation and competency as a specialist until 1916 when the American Board of Ophthalmology was organized. Subsequently other specialty boards were organized and in 1934 the Advisory Board for Medical Specialists was organized. There are now 19 specialty boards actively functioning under the authority of the Advisory Board of Medical Specialists. It is as difficult to fit the available resident trainee to the available residency position as it is to select the proper first

year internship for the recent medical school graduate. However, in 1927 the Council on Medical Education and Hospitals first listed approved residencies in various specialties. The list contained 270 hospitals offering 1699 residencies. In 1938 there were 338 hospitals offering 2038 residencies; in 1940 there were 587 hospitals offering 4394 residencies and 726 fellowships; and in 1957 there were 1259 hospitals offering a total of 31,252 residencies and fellowships.

This method of training doctors and taking care of hospital patients is an excellent one. It is protected by individual initiative and desire and by important national medical organizations. It has resulted in a high quality of postgraduate medical education and a high caliber of professional patient care. It should be constantly improved and protected.

A method of preceptorship training also has been developed for the training of recent medical school graduates and for specialists. Preceptorships always had the disadvantage of being the result of a one-man endeavor. The preceptorships, for the most part, were not well adapted to the care of general hospital patients and were definitely incompatible with the residency training program. In 1956, at the Board of Governors meeting of the American College of Surgeons, preceptorships were reported upon most favorably.

There has been a tremendous effort both within and without the medical profession to furnish good medical care to the general population of the United States in every city, town and sparsely settled area. Hospitals have been built in many locations. In order to induce all hospitals to meet high standards of medical care these institutions formerly were approved by national medical organizations. For this purpose the Joint Commission on the Accreditation of Hospitals was organized in 1952. In 1957 there was a total of 6966 hospitals of all kinds in this country. In the same year there were 6041 hospitals eligible for accreditation and 3441 hospitals approved by the Joint Commission on the Accreditation of Hospitals. In 1957, 854 hospitals were approved for internship and 1259 approved for residencies and fellowships. All hospitals approved by the Joint Commission on the Accreditation of Hospitals are potentially qualified for training interns, residents and fellows.

Since there are not enough physicians seeking hospital training to fill the positions now available and approved it is evident that all hospitals cannot become training centers for young doctors despite the facilities of the hospital and the ability of the professional staff. Nevertheless, new hospitals will be built and more of them will seek approval for training programs.

Hospitals have developed along with the minds of men and it is well to recognize that postgraduate medical training and professional patient care are two different subjects. They can either be combined or good patient care can be separated from internship and residency training programs. Good postgraduate training usually coincides with good professional patient care. Where professional research and experimentation are carried too far in the training program, patient care may suffer. It is possible to establish a high standard of patient care in a hospital without internship or residency training programs. If all doctors concerned with patient care are properly qualified, the routine patient care may be of an even higher standard than the care administered by a doctor of less experience. When the members of the professional staff of a hospital constantly strive for the best possible patient care they, as a staff, will be constantly teaching themselves. Thus, their training will continue throughout the years.

In order to train specialists in various fields of medicine there must be a sufficient number of patients available for the trainees' care. Fortunately, the economic condition of our country is such that there is no foreseeable future in which there will be an abundance of indigent patients. In the decade from 1910 to 1920 the small number of people covered by hospital insurance or insurance to pay the physician or surgeon fees was hardly worth considering. There are no figures available for this period. The Baylor University Hospital Plan (1929) is generally credited with originating the concepts of the Blue Cross Plans. In 1933 there were 2000 Blue Cross members in the United States. In 1940, 12,312,000 people were covered by hospital insurance and 8,350,000 covered for medical and surgical expenses. In 1957 there were 121,432,000 people covered by insurance paying hospital expenses, 108,931,000 with insurance covering surgical expenses, and 71,813,000 insured for medical expenses. Those people with insurance paying surgical and/or medical expense usually have the right to select their own doctor. In order to furnish enough patients for the hospital resident to

gain the necessary experience it is not uncommon for the patient with insurance benefits to be cared for by a trainee rather than by a qualified doctor. With the tremendous increase in the number of people insured for hospital and medical or surgical expenses, it becomes obvious that there will be a continuing decrease in the number of patients available for care by the resident staff.

The approved internship and residency training programs carried out in the good private hospitals are expensive. In Baltimore, calculations from four private general hospitals ranging from 194 to 362 beds showed a cost according to bed capacity from $113,000.00 to $220,000.00 per year. When considered from the standpoint of the number of house officers, the range was $4,362.00 to $5,641.00 per house officer, or an average of $5,282.00 per year per house officer. In spite of the willingness to spend this amount of money there are not sufficient physicians to fill the available house officer positions and not enough indigent or insurance patients upon whom these house officers can train. Throughout the country there is a desperate attempt to fill the house officer vacancies. The field becomes highly competitive and increasingly costly. In order to relieve the shortage of physicians desiring training it has become necessary to resort to the use of many poorly qualified foreign medical school graduates who, in addition to being deficient in basic medical school training, cannot adequately speak or understand the English language. The question arises as to why hospitals should struggle to carry on an internship and residency program when in reality there are not enough doctors seeking this training to fill the training positions offered at this time.

If a clear distinction is made between a hospital that offers internship and residency programs and a hospital that is qualified and approved for the best possible patient care, every hospital in this country not interested in a house officer training program can become a center where professional service of a high caliber can be rendered to the patient. It should be, and undoubtedly is, the goal of the medical profession to render to hospital patients everywhere the highest standard of professional treatment. This can be accomplished if a method of manning hospitals without interns or residents is carefully considered and planned and if the method is then classified and approved by a national medical organization or a combination of national medical organizations.

A method of manning hospitals without house officers should include a carefully considered use of qualified physicians not now utilized in the hospitals. There should be an endeavor to unite the nursing profession by absorbing the practical nurse. With a united single nursing group there could be provided all necessary levels of training for the capable nurse. The goal for the nurse could be elevated to include such duties as assistant to the operating surgeons or assistant to the ward doctor and many other desirable goals with remuneration sufficient to make them attractive. It would be well to have such a program drawn up by a committee of prominent physicians selected from various branches of the profession and representing all the localities of this country. This does not indicate the abandonment of teaching. As a matter of fact, it shifts the emphasis from the well protected 1259 hospitals with house officer training programs to the 5707 hospitals without such programs. It assures continued teaching since any professional medical hospital staff that strives continuously for better patient care will be constantly teaching itself. Such a program probably would cost less in money, time and effort, and thus the surplus of each could be expended in patient care. There would be for each hospital in the United States a goal that could be attained. Instead of being considered second-rate because they have no interns or residents, these hospitals could look with pride and distinction upon their qualification by the approving organization. Approval would indicate that they were furnishing an exceptionally high grade of medical and surgical care to patients admitted to their hospitals and that the hospitals were carrying on a continuous teaching program for their medical staffs. Such a hospital program would be useful anywhere in the United States, in the large city as well as in the hamlet.

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Book Reviews

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Atlas of Technics in Surgery. By JOHN L. MADDEN. M.D., F.A.C.S., Appleton-Century-Crofts, New York, 1958.

This work is a large atlas-type reference book for those who desire a complete verbal description of all the commonly performed operations and many less commonly performed surgical procedures and techniques. The atlas is composed as follows:

1. Large anatomic illustrations which are properly and adequately oriented and sufficient in number to allow understandable progression through various surgical procedures.

2. Adequate and understandable but not verbose descriptions detailing the various progressive steps in the depicted operations on adjacent page (thus obviating page flipping).

3. Critiques, comments, theoretic considerations and general evaluation of the procedures by accepted authorities.

4. Bibliography of textbooks and journals at the end of each surgical description.

This atlas is especially suited for those in surgical training and those adequately trained surgeons who find themselves in need of a refamiliarization with the infrequently performed operation. Included also are many of the "newer" vascular, cardiac and shunting procedures.

In summary, I feel that this atlas would be an asset to any surgeon's library with the admonition that the operations depicted are overly simplified and bloodless, *i.e.*, this is not a surgical cookbook but an atlas.

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The Recovery Room: A Symposium. By J. ADRIANI, M.D., and J. B. PARMLEY, M.D. 123 pp., Charles C Thomas, Springfield, Ill., 1958.

The authors of this small volume have attempted by means of a question and answer technique to determine the factors that contribute to an efficiently operated recovery room. The diversified opinions of 45 authorities from the fields of Anesthesia, Surgery, Nursing and Hospital Administration are presented in an effort to view all sides of the problem as it might apply to the recovery room in private, tax-supported and military hospitals. The series of questions presented by the authors is exhaustive and covers all of the important aspects of the recovery room with adequate emphasis on construction, administration, personnel and economics as well as the specific problems involved in patient management. Post-anesthetic rooms, recovery rooms, and intensive therapy rooms are carefully differentiated by the authors at the beginning of the symposium, yet considerable overlapping of these three concepts occurs throughout the rest of the presentation. The sections that deal with equipment, general patient management, and the management of special problems are excellent.

Although the question and answer technique does much to introduce the personal flavor of the symposium the over-all effect is a "choppy" one. Some facets of recovery room management on which there is a considerable diversity of opinion are answered by a single individual whereas some points of general agreement are given a number of answers.

Despite these minor objections I believe that this little volume will serve a very useful function for those individuals from the fields of Anesthesia, Surgery, Nursing and Hospital Administration who are faced with the problems of setting up a recovery room or ensuring a smoothly functioning recovery area once it has been set up. The chapters dealing with equipment and the management of general and special problems will be of particular use to the personnel working in the recovery room.

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Roentgen Diagnosis of Abdominal Tumors in Childhood. By CHARLES M. NICE, JR., M.D., PH.D., ALEXANDER R. MARGULIS, M.D., and LEO G. RIGLER, M.D. 75 pp., 52 illus., Charles C Thomas, Springfield, Ill., 1957.

This is a short and concise book on abdominal tumors as seen in childhood, written by three radiologists from the Department of Radiology, University of Minnesota Medical School. Anatomic and embryologic comments with appropriate drawings are presented to better understand the origin and location of masses in the abdomen. The authors strongly emphasize the simple radiologic examination that often helps in arriving at a diagnosis. One chapter is devoted to methods of examination; another chapter deals

with roentgenologic classification; four additional chapters are devoted to masses in various organs, the peritoneal cavity and retroperitoneal space.

This book is well written and concise with excellent illustrations. Radiologists, pediatricians and surgeons as well as students will find it of interest.

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Fifty Years of Neurosurgery. By ERNEST SACHS, M.D. Vantage Press, Inc., New York, 1958.

Dr. Sachs' delightfully intimate account of his career spans the first decades of neurologic surgery. One among the small group of illustrious men whose names are now synonomous with the field, his story reveals the pioneering spirit and dedication that eventually brought international acceptance to this surgical specialty.

In addition to penning professional memoirs, Dr. Sachs has included glimpses of life during the late nineteenth and early twentieth centuries, both here and abroad. Such accounts, as well as those regarding his loved ones, reveal an emotionally warm quality that endears the author to all—even those unrelated to the field of neurosurgery.

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